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The Halford Oration.[1]

THE DEPARTMENTS OF MEDICINE AND SURGERY IN THE UNIVERSITY OF MELBOURNE.

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YOUR EXCELLENCY, LADIES AND GENTLEMEN: I invite you tonight to consider a matter which would have been of special interest to the man in whose honour this oration is established. Professor George Britton Halford held his first class in the medical school in Melbourne in May, 1863. Ninety years ago this solitary professor of anatomy, physiology and pathology commenced to prepare students for their responsible work as practitioners of medicine in this young country. Since Professor W. A. Osborne gave the first Halford Oration in 1928, seventeen orators have described the many attributes of the first professor in our medical school.

It is of interest that Halford, at the time of his selection for the only chair in the medical school of Melbourne, was professor of medicine in the Grosvenor Place School of Medicine, London. His selectors, Dr. (afterwards Sir) James Paget, a household name in pathology and surgery today, and Professor (afterwards Sir) Richard Owen, the great comparative anatomist, showed remarkable prescience in choosing a professor of medicine, and one who had practised actively, to undertake the training of students in anatomy, physiology and pathology, those fundamental disciplines on which medicine, surgery and obstetrics are based. The financial difficulties, ever present in this University, were such at that time that cuts were made in the expenditure on the grounds of the University, and even reductions in the salaries of lecturers in civil engineering and law, "an arrangement which, being called for by the interests of the University, those gentlemen cheerfully acquiesced in" (Scott, 1938). The generosity of the young lecturers and the sagacity of the councillors of Halford's day are worth recording. Such action could not be expected today, since the salaries of the present professors and lecturers at this University are proportionately much lower than they were ninety years ago.

Professor Halford had working with him a staff of six lecturers who were practising doctors: Dr. John Macadam, chemistry; Dr. Richard Eades, materia medica, therapeutics and medical botany; Dr. Edward Barker, surgery; Dr.

[1] The nineteenth Halford Oration, delivered at the University of Melbourne on August 4, 1953.

James Robertson, theory and practice of medicine; Dr. Richard Thomas Tracey, obstetric medicine, and diseases of women and children; Dr. James Edward Neild, forensic medicine. These men were all graduates of overseas universities, and it was not for eighteen years that the first Melbourne graduate, Harry Brooks Allen, was appointed as demonstrator in anatomy. He later added much lustre to the Melbourne medical school as professor of pathology and dean of the school.

There were two graduates in medicine in 1867—Dr. Rees and Dr. Maloney; contrast that duet with the 180 in 1952.

The University of Melbourne is a State University, the Medical School is a Victorian institution. It is fitting that the public of Victoria should be informed of the development of the Medical School in the University of Melbourne, and of the recent decision of the Council of the University regarding professors of medicine and surgery.

The medical course has been lengthened by only one year since 1863. The pre-medical year is devoted to physics, chemistry, zoology and botany. For the two following years the student studies normal human anatomy, physiology and biochemistry. He then proceeds to a teaching hospital, where he spends the next three years. During this period he is taught pathology and microbiology in University lectures and by demonstrations at the hospitals. He spends some time at special hospitals, such as the Women's Hospital and the Children's Hospital, and attends lectures in special subjects. For the greater part of the last three years of the course he is engaged in clinical practice at the general hospital.

In the final examinations the subjects which must be passed separately are medicine, surgery, obstetrics and gynæcology. He graduates with the degrees of bachelor of medicine and bachelor of surgery, and is regarded as being equipped for general practice. Experience and further training will fit him for specialized work. The aim of teaching has been to produce a sound general practitioner. In this the school has succeeded.

The content of the medical course has altered since Halford's day. The preliminary education of a doctor included Greek, Latin and mathematics. Now there is a tendency to replace these subjects by physics and chemistry, and even to encourage specialization at a tender age in the schools. Many experienced university teachers doubt the wisdom of directing the student's mind too early to the scientific studies at the expense of the humanities, and they would prefer to give the requisite instruction in these subjects in the pre-medical course.

Biochemistry has become more important, and as knowledge increases, this subject is bound to occupy a good deal of the student's time in the early years of the medical course. However, much of the *materia medica* and botany which loomed large in the bygone days and required a lot of time for memorizing, has been relegated to the manufacturing chemists. Drugs are scientifically prepared and tested before being placed at the disposal of the doctor. Detailed lists of diets, as in diseases such as diabetes, have been simplified, and the rationalization of symptomatology and treatment has been put on a sounder basis by the discovery of insulin.

While the student has many more subjects to study, the scientific method has brought order out of chaos, and compensated for the expanded curriculum to some extent.

From small beginnings in 1863, the medical school of the University of Melbourne has trained many hundreds of medical men, who have served the people of this State and have given of their best in peace and war. Many have made their mark overseas as practising doctors, research workers and teachers. The system has been the same as obtained in Halford's day—that is to say, a small nucleus of professors at the University teaching the preclinical subjects, and a large number of practising doctors in Melbourne who have undertaken the clinical teaching in the subjects of medicine, surgery, obstetrics and gynæcology, pædiatrics *et cetera*. The one exception has been the appointment of a professor of obstetrics in October, 1929, and the expansion of this chair to include gynæcology in March, 1951.

It is wise to trace the developments in medical teaching elsewhere, before criticising our University too harshly in this regard. The system established by Halford was that generally approved at the time, and one that has proved successful, to judge by the standard of graduates in medicine which this University has produced. In fact, it must be admitted today that the best teachers of medicine and surgery for the students in the later years of the medical course are practising doctors of high repute and professional qualifications, who are selected by the committees of recognized teaching hospitals in collaboration with representatives of the University. The hospitals recognized as general teaching schools for the later years of the medical course for this University are, in order of their recognition, the Royal Melbourne Hospital, the Alfred Hospital, Saint Vincent's Hospital, Prince Henry's Hospital. An advisory board or electoral college, on which the University is represented, recommends to the committees of management (or the Mother Rectress at Saint Vincent's Hospital) the names of doctors suitable for appointment as honorary physicians and surgeons. This ensures, not only that the hospital in question has a high standard of academically qualified men, but that the latter will be suitable teachers on behalf of this University in the subjects of medicine and surgery. Such men become automatically the accredited teachers in those subjects.

The setting up of these electoral colleges is a comparatively recent development. In the early part of this century, public hospitals selected honorary physicians and surgeons by popular vote, having little regard for their academic attainments or their teaching ability. The teaching of medicine and surgery was easy in the days of a few students and one hospital—the old Melbourne Hospital. Some didactic lectures, usually 40 or 50 in number, in both medicine and surgery, were given by some of the senior doctors; the bedside tuition was given by the doctors to whose clinics the students were attached. The students were required to take histories, to examine patients allotted to them, to attend operations and post-mortem examinations, and generally to trace their patient from the time of his entrance to his exit from the hospital.

As the medical course became more elaborate and the number of students increased, and as the number of teaching hospitals multiplied, a senior physician and a senior surgeon were appointed Stewart Lecturers. The duty of such officers of the University was to arrange the curriculum in their subject (medicine or surgery), to recommend to the Faculty appropriate lecturers for the didactic lectures, and to organize the examinations at the end of the final year and act as chairman of examiners, and, as well as that, to organize the diplomas and higher degrees involving their subjects. They have the responsibilities of heads of departments, and control from the University end the duties of a large number of clinical demonstrators and clinical supervisors attached to the general teaching hospitals.

In British and American medical schools these officers would have been given the title and status of professor.

For a variety of reasons the coordination of teaching in the several teaching hospitals has become more and more urgent. In 1946, the late Sir Alan Newton advised the Council of this University to establish a department of clinical studies as a preliminary to the establishment of chairs in medicine and surgery. Sir Alan Newton was appointed director of this department, and during his term of office did much to stimulate enthusiasm in clinical teaching. He was instrumental in having young men appointed and paid by the University as clinical supervisors in the teaching hospitals. He also instituted a record system, whereby the clinical work of the student could be assessed and considered at the time of the final examination. He arranged inter-hospital clinics and an exchange of teachers. He organized regular monthly meetings of a Coordinating Committee of Clinical Studies. At these meetings the deans of the teaching hospitals

(members of the honorary medical staff elected to that office), the Stewart Lecturers and the Dean of the Faculty of Medicine meet to discuss matters relating to the more effective teaching of the students in the clinical years.

The teaching hospitals in Melbourne have developed on the English model. The early clinical teachers came from the Mother Country. The curriculum has been in line with that in Great Britain. The course, first fixed by Halford at five years even when in England the course was shorter, has become standardized throughout the British countries. Only thus could Melbourne graduates enjoy reciprocity of registration. The curriculum in the London schools of medicine—Saint Bartholomew's Hospital, Guy's Hospital *et cetera*—has embraced the pre-clinical subjects as well as the bedside teaching, the University of London being the examining body. Thus there has arisen a local tradition. This same tradition has been developing in the Melbourne teaching hospitals, even though the pre-clinical subjects are taught at the University. It is what Sir Alan Newton in a famous address once called "The Spirit of the Place". He was referring to the Royal Melbourne Hospital and its students. The same applies to the Alfred Hospital and to Saint Vincent's Hospital. This local tradition is valuable. Just in the same way as local government has much to commend it, so has a local pride in one's teaching school, local interest in the human side—the patients, staff and fellow students in the intimacy of the ward or operating theatre. "The spirit of the place" should be preserved.

At the same time it should be realized that a loyalty to one's clinical school does not preclude the larger loyalty to one's University. It is not surprising that Australians, with their roots in the older countries and their medical schools planned on British lines, should look to England for a lead. On the other hand, a young nation may be expected to develop its own individuality and not slavishly follow the methods of the old lands.

Halford realized his opportunity when he established the five-year course in Melbourne.

The Coordinating Committee of Clinical Studies has served a very useful purpose in cementing the bonds between the different clinical schools. There is some diversity of method, and this is advantageous. An experiment in teaching can be tried in one school and the results observed by the others. A free exchange of ideas is permitted, and yet a certain healthy rivalry is retained. Inter-hospital clinics are regularly held, and this helps to bring the students of all the schools in contact at some stage with all the teachers. It would appear that the Melbourne Medical School has reached a stage in its evolution at which it is appropriate, even imperative, to appoint professorial heads of the departments of medicine and surgery. As there are three larger general teaching hospitals, and it is highly desirable to have University representation in each, a third professor should also be appointed.

Since Sir Alan Newton's death, the position of director of clinical studies has been abolished by decision of the University Council, and the Coordinating Committee of Clinical Studies elected the Stewart Lecturer in Medicine, Dr. John Hayden, as its chairman for this year. It will be seen that in the Medical School this University has made a little progress since Halford's time. In fact, there are departments of medicine and surgery, spending £12,000 a year, but with no heads comparable with the professors in other departments such as obstetrics and gynæcology, anatomy *et cetera*.

On July 3, 1950, the Council of this University approved in principle of a chair in medicine and a chair in surgery, and asked the Vice-Chancellor to inquire into the possibility of obtaining finance. In 1951, the Council decided to appoint, under chairmanship of the Vice-Chancellor, a Conjoint Committee having representatives of the Council and of the committees of management of the teaching hospitals and also of the Faculty of Medicine, to advise on the question of chairs in medicine and surgery. As a result of much deliberation and the work of subcommittees of this body, a resolution was passed on November 14, 1952, unanimously making the following recommendation to the Council:

> The Conjoint Committee recommends to Council that the best development of teaching and research in medicine and surgery in the University of Melbourne will be accomplished by the appointment of professors in each of these subjects. The formation of the Departments of Medicine and Surgery should be undertaken in two stages.
>
> *Stage* 1 would be concerned with the appointment by the University of three Professors (two in Medicine and one in Surgery) one professor to be located at each of the Royal Melbourne, Alfred and St. Vincent's Hospitals. These appointments should be made simultaneously, provided:
>
> (i) that if financial provision be difficult one chair might be filled in each of three successive years;
>
> (ii) that candidates of suitable qualifications are available.
>
> *Stage* 2—When this initial stage is complete and each unit functioning satisfactorily, three further professorial appointments should be made as soon as adequate provision is available so that each of the three hospitals would house a professor in each subject.
>
> The Committee further recommends to Council:
>
> (*a*) In each department (i.e., in medicine and in surgery) the University Council shall appoint among the Professors, one professor who shall be head of that department and such appointment shall be made quinquennially.
>
> (*b*) Whenever the above resolutions are in conflict with previous resolutions passed concerning the Chairs of Medicine and of Surgery these shall be the ruling resolutions.

The Council resolved, at a meeting on December 15, 1952, that with the exception of paragraph 6—relating to finance—the recommendation of the Conjoint Committee be approved, and the question of finance be referred to the Finance Committee of the Council. The appointment of professors then depends on finance, and the money is not in sight.

It is of interest to note that a previous committee, in 1949, under the chairmanship of Dr. Leslie Hurley, had recommended that university departments of medicine and surgery be established in the three teaching hospitals above mentioned, but that for the time being associate professors be appointed—and not until at least one year after that system was in full operation were professors of full status to be appointed. This was a decision which at least had the merit that all the then three general teaching hospitals were in agreement. The implementing of that recommendation proved difficult for the University, there being no administrative head of medicine and surgery at professorial level. Hence the reference of the matter to the Conjoint Committee.

Many recommendations have been made in the past years on this issue. Professor R. J. Berry urged the establishment of a medical centre twenty-five years ago. Sir Victor Hurley reported similarly, and strongly advocated the establishment of chairs in medicine and surgery sixteen years ago. Sir Alan Newton submitted a report to the Council on the matter in 1947, and drew up detailed reports of the requirements of such departments. The Department of Clinical Studies with its director was regarded by him as an interim arrangement pending the establishment of the full chairs.

The reason for procrastination is easily understood when it is realized how the three general teaching hospitals have grown up as affiliated with the University—lending their patients, as it were, and providing facilities for the students—while being outside the University's jurisdiction. It says much for the patience, forbearance and statesmanship of the members of the various committees with diverse representation, that at last a unanimous decision has been reached. It only remains for the University Council to act on its decisions and make the appointments.

Surgeons were bound to admit that medicine should be represented academically at first more strongly than surgery, and that if there were to be three professors, one

in each of the three older general teaching hospitals, then two should be in the department of medicine.

Medicine has long enjoyed an academic respectability in the older universities of Europe, and the cynic might suggest that it is not so long since surgery emerged from its primitive "barbarism". Surgery is popularly regarded as some form of cutting, and indeed the early surgeons did all manner of cutting, including that of hair and nails. They were called in by physicians to cut veins and let blood, to cut for stone, to amputate limbs. With the development of the science of descriptive anatomy, surgeons enlarged their domain; but it was not until the discoveries of a chemist, Pasteur, that it became possible, under the enlightened teaching of Lister, for surgeons to extend their procedures into many parts of the body.

Thinking surgeons make no exaggerated claims for the superiority of their craft. A watchmaker does finer work, but he can throw away or melt down a part on which an error was perpetrated—not so the surgeon. The human element will always be paramount. Hence surgeons must be good doctors in the accepted lay sense. The teaching of surgery to undergraduates cannot be divorced from the teaching of medicine. The two go hand in hand. The modern surgeon is not just a horse leech or barber, but a practitioner of medicine who, by aptitude and special training, is able to undertake operative or manipulative procedures for the diagnosis of the disease and the treatment of the patient. Complicated surgical technique is best learnt by apprenticeship after graduation.

Surgery has contributed to a more detailed knowledge of the course of disease. The observant physician learns living pathology at the operating table. The surgeon, on the other hand, obtains his best results when acting in close collaboration with the physician or anæsthetist, who patiently questions, critically examines and employs every necessary modern diagnostic or anæsthetic aid.

Carbuncles, boils and extensive cellulitis, which formerly required the knife, are now frequently cleared up with injections of antibiotics. Middle-ear and mastoid infection, which used to be treated by operation, are often similarly responsive to medical measures. Conversely, surgical operations are performed for diseases of the large bowel, such as ulcerative colitis, some toxic goitres, and peptic ulcers which do not respond to medical treatment. Diseases of the heart which were regarded as the field of the physician are now being attacked by the surgeon.

Medicine and surgery are thus two facets of the single science. The volume of knowledge has been so enlarged that it is no longer possible to speak of a surgeon as a physician who operates, or of a physician as the brain behind the surgeon's hands. Specialization has become a necessity.

In recent times so many advances have been made that diseases which twenty years ago were amenable only to treatment by surgery are now treated by medicine, and diseases which were regarded as properly those to be observed to the end by the physician are now cured by the surgeon. Diagnosis is permanent—smallpox as in the time of Hippocrates is the same today. Treatment, whether medical or surgical, is subject to change.

These reflections bring me to the main theme of this lecture. I am not competent to discuss in detail the qualifications required of a professor of medicine, but I may be permitted some observations on the special attributes of some of the heads of departments of surgery, and the functions of such departments which I observed during a recent visit overseas.

In 1950, the Nuffield Foundation in Great Britain provided a grant in aid, and by many introductions enabled me to see at first hand the work in the British university departments of surgery. The Rockefeller Foundation did the same for me in the United States of America and in Canada. Some of the substance of those remarks has already appeared in a report to the Council of this University (March, 1951).

It may be held that the system of teaching surgery to undergraduates here has proved successful, and therefore it is unwise to change it. It has been particularly suitable for Australian conditions. A practical subject should be taught by practical men who are daily engaged in the practice of the art. This is a policy in other university departments such as law, and it is not proposed, under the new régime, to alter that system.

The old and well-tried method of apprenticeship of senior students to teaching physicians and surgeons will continue. Recently, a return to the older system has developed in this medical school, in that senior students are allotted to general practitioners for a week or two, in order that they (the students) may study illness in the home. There is thus no tendency to depart from time-honoured methods in the Melbourne Medical School.

The volume of knowledge in the pre-clinical subjects—anatomy, physiology, biochemistry, microbiology, pathology—has so increased that professors and teachers in these subjects must necessarily commence to specialize early. The very nature of their speciality, involving mainly laboratory work, tends to shift their interest from the patient to his parts. This is not a fact to be deplored, but is an inevitable consequence of the development of the ancillary sciences, which have contributed so much, and will contribute more, to medicine and surgery. In this medical school at least two of our pre-clinical professors have had experience as clinical teachers. This is in the Halford tradition.

The absence of professors of medicine and surgery in our school has not been unimportant internationally. No general teaching hospital in Melbourne is considered by the American authorities to be of the first grade. The reason given is that there are no professors of medicine and surgery. This is a handicap to our clinical schools, which enjoy prestige in other respects. It prevents an interchange of our best teachers, and reflects on our standards to the detriment of our graduates when they go overseas.

Professors of medicine and surgery are the links between the laboratory worker, the researcher, the student and the practitioner. Discoveries are made, isolated facts are ascertained. The integration of these new discoveries into the art and science of medicine and surgery is the function of the professors. They have to keep their subject up to date. Original research may be done by them or their assistants, but their first important work is integration. For this work many are called, but few are chosen.

In a report to the Carnegie Foundation (on medical education in the United States and Canada), Abraham Flexner in 1909 criticized the teaching of medicine and surgery in the United States of America and Europe. There was a lack of scientific spirit in the hospital wards and classrooms—too little inquiry and investigation, and too much vain repetition of established dogmas. Many of the mushroom-like medical colleges which had sprung up of necessity in the rapidly expanding areas had become second-rate workshops, having little or no affiliation with universities. The light of learning was growing dim. A closer link with the laboratory, a different type of teacher—that is, one grounded in the scientific method, having a close association with the university and with men interested in the fundamental sciences—was urged. Stress was laid on the need for inquiry, investigation and critical analysis.

The great American and Canadian medical schools adopted these recommendations. They established departments of medicine and surgery, the professorial heads of which were specially selected doctors with a flair for teaching and inspiring students in investigations of disease and newer methods of treatment. Johns Hopkins Hospital, Baltimore, had been held up as a model.

The Scottish universities have for long recognized medicine and surgery as worthy of chairs. In 1772, Thomas Simson was appointed Chandos Professor of Medicine at Saint Andrews. A chair of surgery was founded in Edinburgh in 1831. The first incumbent was Dr. John William

Turner. He was succeeded in 1836 by Sir Charles Bell, who had made a great reputation as a scientist and anatomist in London. Bell's palsy (paralysis of the seventh cranial nerve) perpetuates his name.

Joseph Lister, later Lord Lister, was appointed Professor of Surgery in Glasgow in 1860. There he carried out his researches on inflammation and antiseptics. He practised privately, taught students and investigated—and with what benefits for mankind! His old school in London was slow to appreciate him, but today there is a memorial plaque in the ward in which he served as a dresser and house surgeon. Later, as Professor of Surgery in Edinburgh and then in London, he performed that miracle which rightly divides the science and art of surgery into two eras—before and after Lister.

It may be argued that Lister would have done his work anyhow, whether or not he had been made a professor of surgery. This is open to question, for he received not a little discouragement, and even obstacles were put in his way by his surgeon contemporaries, who ridiculed the use of the microscope by one of their craft. At the time of his appointment as professor, the Board of the Glasgow Infirmary was reluctant to appoint him to its staff—stating that its institution was a curative, not an educative one.

How times and manners have changed! Glasgow has now a splendid department of surgery with full-time professors, and opportunities are provided for young men with ideas to try them out.

Lister's original experiments were carried out on animals, mice and small creatures. Only after his methods had been thoroughly tested did he apply the results to a human being.

It is important for lay people to realize that in our large teaching hospitals only well-tried methods are adopted. Experimental work which may save a life is not easy to embark upon without the scientific control and the judicial appraisement of result which are the proper functions of university departments or clinical research units.

The Function of Universities.

In his address at Newcastle, Sir William Osler endeavoured to answer the questions: What is university work? What is a hospital unit? What connexion have they with each other? What interest have they for us and for the community at large?

> All are agreed that a University has a dual function—to learn and to advance learning. In mind, manners and morals the young man seeks life's equipment when he says to his Alma Mater in the words of the Psalmist: "O learn me true understanding and knowledge". . . . To learn the use of his mind, to learn good manners, and to learn to drive Plato's horses, form the marrow of an education. . . .

University education should be comprehensive, fitting a man, in Milton's words, "to perform all the offices, private and public, of peace and war". The other great function of a university is to advance learning, to increase man's knowledge of man and of Nature.

As a part of the university, the medical school has two functions: teaching—that is, passing on the knowledge already tested and confirmed—in fact, medical orthodoxy—and secondly, adding to that knowledge—that is, research. It is not given to all to be brilliant researchers, nor is it likely that discoveries can be made according to the calendar or the clock. Nevertheless, it is desirable that students should be brought up in an atmosphere of critical inquiry, and be made aware of the growing edge of the subjects they study. In their post-graduate years they are thus stimulated to continue their studies, watch the unknown being gradually conquered, and apply advanced knowledge for the benefit of their patients.

The first function has been teaching in this school, and of the professors in the pre-clinical subjects in the bygone days it can be claimed that they taught well—they laid the foundations for some leading world investigators in the field of medicine. By their fruits they are known.

There is a place for the pure researcher in the medical field. To quote Osler again:

> A few exceptional men . . . are really too good to teach; but for the majority, daily contact with students, and a little of the routine of teaching keeps us in touch with the common clay, and are the best preservatives against that staleness so apt to come as a blight upon the pure researcher.

In some of the heavily endowed medical schools of the United States of America, such as the Johns Hopkins, a professor of surgery is sought by interview, invitation and negotiation. One who has already shown an aptitude for research or for opening up a new branch of surgical knowledge is sought. Examples of distinguished professors of surgery in that university are the late Dr. Harvey Cushing, the father of modern neurosurgery, and the present holder of the chair, Professor A. Blalock, who has done so much for the surgery of the vascular system. The professor, who is a strictly full-time officer, is assisted by a full-time assistant professor, who relieves his chief of much of the administration of teaching of undergraduates. There are also professors and associate professors in specialist branches of surgery. Most of them work part-time, practising their profession and earning most of their income in that way. They devote set hours—say each half-day—to their university work in the hospital, just as many of our honorary physicians and surgeons do. The professor-in-charge is on a salary. If he operates on private patients, the fees received go into a pool for the benefit of the department. This system is favoured by seven private and one State university medical schools, but exceptions exist as at Northwestern University, Chicago, where the professors and staff derive their main income from private patients in and adjacent to the teaching hospital.

Intermediate between the two above mentioned is the geographical full-time professor who works only at his teaching hospital, but may earn up to the value of his salary from private patients examined and treated only at the teaching hospital. This is the most widely used full-time plan in American university medical schools today. The financial advantages are obvious.

Geographical full-time was introduced by Harvard University and the Peter Bent Brigham Hospital in 1913. The professor and some of his associates were provided with offices in the hospital. They were privileged to carry out private practice in the institution and collect their own fees. The only limit placed on private practice is the broad statement: "Private work is not to interfere with University duties."

From my observation, the Harvard professors filled their days with university work, but the President of the United States had the services of one of them as his personal surgeon. They do not abuse their privilege.

Although the strict full-time system has improved the quality and quantity of basic research in departments of American universities, it has been adopted by few of those universities in recent years. This is related to increasing costs.

At the University of Chicago the members of the clinical faculty are appointed on a full-time group practice basis and render service to paying and non-paying patients. All fees for professional services are collected by the University to go into a common fund to be used for the best interests of the clinical departments.

This system is possible because the hospital is a University hospital. There is no reason why this system should not function in Melbourne.

In England, almost all the London and provincial universities have full-time professors in medicine and surgery. Leeds still retains a part-time professor of surgery, and Guy's Hospital, London, has a Director of Surgical Studies, who devotes almost all his time to the duties of his office. The professorial unit developed in the earlier part of this century in London in University College (Lord Lister's old school), and it has been, with a few exceptions and modifications, adopted in the other medical schools of the University of London.

Boston (United States of America) and Toronto (Canada) have been compared with Melbourne. In all three, a university medical school has three or more general teaching hospitals affiliated for purposes of undergraduate tuition in clinical medicine and surgery. Toronto has a professor of surgery located near the large Toronto General Hospital—that is, in the Banting Institute across the road. He controls all the public beds in the teaching hospitals, and associate professors work under him at the other teaching hospitals. At Harvard, in Boston, there are three professors of surgery—one at Massachusetts General Hospital, one at Boston City Hospital and one at Peter Bent Brigham Hospital. They are full-time professors, one of whom is the recognized University head of the Department of Surgery.

Australian experience in full-time chairs in medicine and surgery has been limited to the University of Sydney. Part-time chairs have been the rule in Brisbane until this year, when a full-time professor of medicine was appointed, and recently Adelaide appointed a full-time professor of medicine. Already departments of clinical research are in existence in our teaching hospitals; the main emphasis is on medicine. The chairs in medicine and surgery can and should be made attractive. It is the men that matter.

The full-time professors in medicine and surgery, unencumbered with the worries of private practice, if any, outside their hospitals, and secure in their appointment for years, will be able to devote their time and energy to the organization of teaching in their subjects, know their students personally, inspire them, direct them and influence their future practice as do our pre-clinical professors today, and as Halford did in past days. They will investigate where they can most profitably advance the knowledge of their subject. They will play their part in post-graduate medical education, and maybe assist in elevating this long-neglected university activity to its proper status. They will find an enthusiastic group of clinical teachers, experienced in the art, anxious and willing to assist them, to work under their leadership. They will find also a group of young doctors with high ideals and a keenness for investigation who are anxious to be directed in the solution of technical problems. They will find a body of citizens who are alive to the potentialities of their great school, and who will not be ungenerous in their support. What an opportunity!

What advantages have accrued from departments of surgery of recent years? Lord Lister's work has been mentioned; Professor Sir James Learmonth, noted for the investigation of peripheral vascular disease, operated on King George VI. Other well-known names include the following: Boyd of Manchester; Jefferson of Manchester; Cairns of Oxford (neurosurgery); Lambert Rogers (a Melbourne man) of Cardiff; Grey Turner of London and Newcastle; Wangensteen of Minneapolis, a designer of apparatus which has saved many lives; Blalock and Cushing of Baltimore—mentioned earlier; Moniz of Lisbon; Schmitzu of Tokyo; Leriche of Paris; Kirschner of Berlin; Dubouillet, Glenn and Humphreys of New York; Ravdin of Philadelphia; Churchill, Smithwick and Moore of Boston; Gallie and Janes of Toronto; Coller of Ann Arbor; Penfield of Montreal. All these contemporary professors of surgery have contributed in our day to the development of the art and science of surgery, and influenced the whole field of medicine.

Melbourne, in more leisurely days, was the home of Hamilton Russell, whose name is widely remembered in several branches of surgery. Though never called a professor, he was literally a true disciple of Lister. This is the answer to those who are slow to appreciate the modern trend for university departments of surgery. I have authentic information that great foundations in England and the United States of America are watching with interest our activities in this direction.

Conclusion.

It has been shown that the University Council has approved of the appointment of professors of medicine and surgery as outlined above—subject to finance being available.

The financial difficulties are not insuperable. An Appeal Committee has been formed for the purpose of raising money for the endowment of the three chairs. The money raised would enable salaries to be paid for a few years, and help in the provision of equipment. Some money for the maintenance of the chairs could easily be provided by arrangement with the committee of management of the teaching hospitals.

The leaders in the profession and the teaching staffs of the hospitals will benefit by close association with the professorial departments. The medical profession generally has derived much from the predecessors of the professors, and the future members of the profession will owe something to their teachers. It is not unreasonable to request financial aid from these professional men.

It is possible that some financial help may be provided by governments. It only remains for the finance committees to use a little imagination and initiative to devise ways and means of finding money from the resources available to support the departments of medicine and surgery. The University Finance Committee might set up appropriate machinery if it deemed it necessary.

However, the earning of fees is not the first duty of professors, but is only incidental. If a real advance is to be made, the heads of departments must be freed from the necessity of running a profitable business, and allowed time to meditate and to use their brains to the best advantage.

In Great Britain the National Health Service pays salaries to all doctors in public hospitals, and professors of medicine and surgery participate in the scheme. It is appropriate that the staff of departments of medicine and surgery in this University should be included in the medical benefits service of this country. The staff of other departments in the medical school which renders a service to patients in so-called public hospitals could be similarly reimbursed.

The salaries paid to professors must be in keeping with their professional status. A parsimonious policy engenders bitterness and a justifiable resentment. Professors of medicine and surgery will not have a life of cloistered seclusion, but will take their place in the sick room and the operating theatre, as well as in the lecture hall and the laboratory. They will be faced with the emergencies of illness and accident, life and death.

The teaching hospitals have shown a lively interest in the project, and have already made arrangements to accommodate the professors and provide beds for teaching purposes. It is surely not too much to ask that this University, as it approaches its centenary, should accord that rank and status to medicine and surgery which all other universities with medical schools of similar size in the English-speaking world have granted.

There is a tendency to think of the University as some remote body between heaven and earth (some even suggest the place beneath). It is our University, dealing with problems which concern every man, woman and child in this community. The medical school is a part of it, and an important part. But for the assistance of its trainees many of us would not be here tonight. I appeal to the business man, the farmer, the politician, to help us in the prosecution of this scheme.

The public of Victoria has been ever ready to assist a worthy cause. If it considers that the improved training of doctors is important, if it even takes the narrow but quite justifiable view that a longer and happier life is desirable, it will support the appeal for funds to establish the chairs of medicine and surgery. This is a good investment for future generations of Victorians, and one that will pay dividends in the donor's lifetime.

The Melbourne Medical School has a fine tradition. Its graduates have a goodly heritage. This generation has a duty to posterity, and the privilege of completing the superstructure on the foundations so well and truly laid by Professor George Britton Halford.

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HANSEN'S DISEASE IN NEW GUINEA.

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DURING a two months' tenure of a medical studentship under the Health Department in New Guinea at the beginning of 1952, I saw a number of patients suffering from Hansen's disease, both at Gemo Island out of Port Moresby and at the Rabaul Native Hospital in New Britain. It may be interesting to consider a summary of these cases from both a public health and a treatment point of view.

Hansen's disease (leprosy) is a specific infectious disease of man caused by *Mycobacterium lepræ*, and is usually characterized by (i) a long incubation period, (ii) a long course with exacerbations and remissions, (iii) primary involvement of skin, mucous membrane and/or peripheral nervous system.

Although there are no definite statistics available, it would appear from general impressions that Hansen's disease is now more commonly seen in New Guinea and New Britain than previously. At times this disease has been prevalent in Europe, Greece and the United States of America, but recently there has been a great spread in the Pacific islands, where today the highest rates of infection are found—for example, at Nauru Island, 60 cases per 1000 of population. It has been estimated that there are 5,000,000 cases of this disease in the world.

Classification of Hansen's Disease.

There have been many attempts to classify this disease, both on the appearance of the skin lesions and on the microscopic examination of the lesions in various parts of the body; however, the simplest classification, which I will use (South American classification), is based on the microscopic appearance of the lesion. There are three main types, as follows.

The Lepromatous Type.

Microscopic examination of the lesions in the lepromatous type reveals numerous acid-fast bacilli, but there is very little cellular response in the tissue. The typical lesions are symmetrical nodules, either in or on the skin, papules, macules or diffuse infiltrations. The characteristic form is the leproma, which on examination of sections is found to contain lepra cells (macrophages with bacilli and fat droplets). The leproma is found chiefly on the extensor surfaces of the arms, or on the exposed part of the face, in particular the eyebrows, cheeks and ears, and combined with a general thickening of the subcutaneous layer of the nose and lips gives the characteristic leonine facies. The respiratory or the digestive tract may also be involved, the mucous membrane is primarily attacked and the patient may complain of an obstructive nasal discharge, dysphagia, or hoarseness and shortness of breath due to laryngitis and thickening of the vocal cords.

The Tuberculoid Type.

The tuberculoid type also includes the neural type of the Cairo classification.

In this type of the disease the nerves may be affected or there may be typically cutaneous leprids which are often macular and have an irregular asymmetrical distribution. Microscopic examination discloses very few bacilli and a more pronounced cellular reaction than in the lepromatous type. A typical tubercule may be formed with epithelial cells, giant cells, lymphocytes and plasma cells. Clinically, these patients are seen at an earlier stage than those with the lepromatous type. Areas of skin are affected by early hypopigmentation, anæsthesia and anhydrosis. If the nerves are primarily involved the patient will often complain of numbness, tingling and a sensation sometimes likened to burning in the skin. Later the nerves may become thickened and tender and pain may be experienced over the distribution of the nerve. Finally, the nerve may be completely destroyed, with atrophy of the muscles supplied by that nerve, and trophic resorption of the small bones of, most commonly, the hands and feet accompanied by large, painless ulcers.

The tuberculoid cases usually run a chronic course and if recognized early are the best cases for treatment. The nerves most frequently affected are the ulnar nerve just above the elbow, the facial nerve, the lateral popliteal nerve as it passes round the head of the fibula, the radial nerve where it is close to a bone, and the great auricular nerve behind the external jugular vein.

The affected nerve first becomes tender and later thickened. Leprosy is the only disease in which thickening and tenderness appear together in a nerve, so this is a useful diagnostic point, and in all suspected cases the ulnar nerve at least should be palpated.

The Non-Specific Type.

Microscopically, the lesions in the non-specific type are intermediate between those of the other two types. There are only a few bacilli and there is a slight cellular reaction which is limited to the perivascular and perineural tissues. The lesions are not characteristic enough to permit classification.

Distribution.

Although the extent of the various types varies in different geographical areas, in New Guinea, as in most parts of the world, the tuberculoid type predominates.

Diagnosis.

Early diagnosis is very important because of the recent advances made in treatment, but in tropical countries this is made more difficult by the almost universal presence of other skin diseases. However, diagnosis may be safely based on the following four criteria.

1. The age of the patient and any history of exposure to the disease are important. Although the mode of transmission of the bacteria is not known, nearly all patients with proven leprosy give a history of prolonged direct contact beginning in childhood. The resistance to leprosy increases with age, and therefore many early cases are recognized in native children between the ages of ten and fifteen years who have been living in close contact with either parents or relations who are known to have Hansen's disease.

2. Any native who presents with skin or mucous membrane lesions of unknown origin should always be examined for Hansen's disease. The most reliable method is to take a nasal scraping and examine this microscopically for *Mycobacterium lepræ*. Any suspected skin lesions should be scraped and the scrapings examined under the microscope.

3. In a suspected case the skin sensation should be carefully examined for response to light touch, heat and pain.

4. Peripheral nerves known to be commonly involved —for example, the ulnar and radial nerves—should always be examined for the presence of thickening and tenderness.

After these four criteria have been considered, diagnosis can then usually be made; however, in a non-endemic area, leprosy is seldom diagnosed early, but it should always be kept in mind in all native places, particularly amongst the aborigines in the north of Australia, and in relation to seamen and others visiting Australia from endemic areas.

Treatment.

Treatment of Hansen's disease, although more successful in recent years, is still largely experimental. The patients

are segregated to enable them to be put on a strict régime of rest and adequate diet similar to that given to tuberculosis patients, and recently the sulphone group of drugs has been found of value in arresting, and in some cases curing, early Hansen's disease. These drugs are derivatives of diaminodiphenylsulphone, and the most common ones in use are "Promizole" and "Sulphetrone". Some good results have been reported, particularly with early tuberculoid disease. Streptomycin has recently been used in the United States of America, and has proved useful when other methods of treatment have failed.

Conclusion.

Although Hansen's disease is not a public health problem amongst the white population of Australia at present, it has become more prevalent amongst the black Australians (a recent survey showed 56 cases per 1000 in the Northern Territory), and it is important that these cases should be recognized and the condition treated. Occasionally, Hansen's disease is recognized in people visiting this country, and as it has at times in the world's history been an important disease amongst white people, it is vital that it should be recognized in the community and treated early. However, it is now the general opinion that although Hansen's disease may be infectious, it will not be contracted under good hygenic conditions and in communities where the general standard of health is high.

Hansen's disease, like tuberculosis, malaria, yaws and dysentery, is a very great problem in the Pacific islands at present; but with the spread of modern medical knowledge and an adequate medical service amongst the native peoples, it can be controlled with a possibility of eradication in a number of years.

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GALL-STONES IN SEVEN THOUSAND POST-MORTEM EXAMINATIONS.

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THE cases in which gall-stones were found in my 7000 tabulated post-mortem examinations at the Royal Adelaide Hospital between the years 1920 and 1948 are recorded in the *Medical and Scientific Archives of the Royal Adelaide Hospital* (Number 6 for the year 1926 to Number 29 for the year 1949). As well as the sex and age of the subjects, all other pathological lesions of importance are also entered. As 871 of these 7000 persons (about 8%—434 males and 437 females) had gall-stones, considerable information is available for ascertaining the age and sex distributions of gall-stones and for possible evidence of their association or not with other conditions.

As there were nearly twice as many men (approximately 4592) as women (2401), it is clear that gall-stones are about twice as common in women as in men.

In Table I are given the number of persons with gall-stones (and in parentheses the number of autopsies) for each sex at each age group. Figures for the first 3000 autopsies (1920 to 1934) and for the last 4000 (1934 to 1948) are also given. It is probable that not all gall-stones were recorded in the earlier series, and that few were missed in the later one. In spite of this the figures do seem to show that gall-stones have become more frequent between 1920 and 1948. They also show an aging population.

In males, no gall-stones were found in 193 aged under twenty years. A few cases appear in subjects in the twenties and thirties. About one in 11 men in the fifties will have gall-stones, the figure increasing to about 1 in 4+ in the eighties.

Of girls aged under twenty years, one in 136 had gall-stones. Nine affected women in the twenties compare with six men for approximately an equal number examined. There are more gall-stones in women in the thirties than in men in the fifties. One in four women aged between sixty and seventy years will have gall-stones, and over seventy years one in three.

In Tables II and III, the kinds of gall-stones are recorded for the last 4000 autopsies for the sexes separately. The term "solitary" gall-stone comprises the oval type with radiating cholesterol crystals of which there may be more than one.

In women the commonest type of gall-stone is the multiple facetted type, but in men it is surprising to see that the small pigment type is more often found.

It is well to bear in mind the frequency of gall-stones in women aged over thirty years and in men aged over fifty years, and to remember, whatever symptoms the patient may present, that one in eight men in the sixties and one in four in the eighties, and one in five women in the fifties and one in three over the age of seventy years, may be expected to possess gall-stones.

Gall-stones, carcinomata and coronary disease leading to infarction or fibrosis of the cardiac muscle all increase in frequency at about the same ages with advancing years. Is there any possible association between them based perhaps on some nutritional dysfunction? The following data from these post-mortem records seem to show that gall-stones have no special association with carcinomata or with coronary disease.

There were 24 persons with gall-stones among 159 with carcinomata in the sixth thousand autopsies, and 23 with gall-stones among 164 with carcinomata in the seventh thousand—that is, 47 in 323 with carcinomata, or about one in seven. Twenty-six of the subjects with gall-stones were men and 21 women. There were two persons in the forties, nine in the fifties, 21 in the sixties, 13 in the seventies and two in the eighties. Ten with gall-stones in 47 with cancers of the stomach is higher than the average, but the figures are too small for an opinion to be formed on them. The same applies to five with gall-stones in 24 with carcinomata of the prostate and to four in 19 with carcinomata of the breast. The facetted, the solitary and the pigment types of stones were all about equally represented. When the figures are taken all together, the number of persons with gall-stones amongst those with carcinomata are about what would be expected from the age groups concerned. Conversely, of 291 persons with gall-stones, 41 had carcinomata—about one in seven. Of these, eight out of 68 with facetted gall-stones, eight out of 61 with the solitary type, 12 out of 49 with pigment stones, and 13 with "gall-stones" unspecified or mixed, had carcinomata.

Infarction or fibrosis of heart muscle may be taken as indicating the presence of considerable coronary atheroma. In the sixth and seventh series, 51 persons had cardiac infarction with hypertrophied hearts and 39 infarctions only; nine of the former and six of the latter had gall-stones—that is, 15 out of 90 cases. Forty-four had fibrosis of the heart muscle, which was hypertrophied, and five of these had gall-stones, and 47 had fibrosis only, of whom eight had gall-stones—that is, 13 out of 91 cases. Combining these we get 28 with gall-stones in 181 examples of severe coronary disease, or about one in 6·5. Eighteen were men and 10 were women; but cardiac infarction and cardiac fibrosis are both much more common in men. Twenty-six were women among 90 subjects with infarctions, and 15 were women among 91 subjects with fibrosis.

Conversely, nine persons out of 68 with facetted gall-stones, nine out of 61 with the solitary type of gall-stone and 11 out of 110 with "gall-stones" (unspecified or mixed) had infarction or fibrosis of the heart muscle. It is rather

TABLE I.[1]

Series.	Age Group (Years).										Age Not Stated.	Total.
	Under 10.	10 to 19.	20 to 29.	30 to 39.	40 to 49.	50 to 59.	60 to 69.	70 to 79.	80 to 89.	90 and Over.		
Gall-stones of All Kinds in Seven Thousand Autopsies (Male Subjects).												
First three thousand ..	— (13)	— (101)	4 (151)	4 (200)	22 (299)	26 (355)	45 (440)	43 (302)	14 (70)	— (4)	— (70)	158
Last four thousand ..	— (1)	— (78)	2 (137)	4 (187)	11 (304)	55 (573)	91 (708)	83 (459)	30 (121)	— (8)	— (11)	276
Total..	— (14)	— (179)	6 (288)	8 (387)	33 (603)	81 (928)	136 (1148)	126 (761)	44 (191)	— (12)	— (81)	434
Approximate incidence ..				1 in 48	1 in 18	1 in 11	1 in 8	1 in 6	1 in 4+			
Gall-stones of All Kinds in Seven Thousand Autopsies (Female Subjects).												
First three thousand ..	— (18)	— (64)	6 (148)	17 (161)	18 (154)	26 (168)	36 (163)	28 (71)	11 (26)	— (1)	2 (15)	144
Last four thousand ..	— (1)	1 (54)	3 (145)	19 (163)	32 (215)	71 (291)	85 (310)	64 (179)	18 (48)	— (4)	— (2)	293
Total..	— (19)	1 (118)	9 (293)	36 (324)	50 (369)	97 (459)	121 (473)	92 (250)	29 (74)	— (5)	2 (17)	437
Approximate incidence ..				1 in 9	1 in 7	1 in 5—	1 in 4	1 in 3—	1 in 3—			

[1] The total numbers of autopsies at this age are given in parentheses.

TABLE II.

Gall-stones in Fourth, Fifth, Sixth and Seventh Thousand Autopsies (Male Subjects).

Age Group. (Years.)	Number of Autopsies.	Types of Gall-stones.				Total.
		Facetted.	Solitary.	Pigment.	Unspecified and Mixed.	
Under 10	1	—	—	—	—	—
10 to 19	78	—	—	—	—	—
20 to 29	137	—	1	1	—	2
30 to 39	187	—	1	2	1	4
40 to 49	304	2	1	3	5	11
50 to 59	573	15	10	8	22	55
60 to 69	708	11	20	21	39	91
70 to 79	459	21	12	24	26	83
80 to 89	121	6	4	11	9	30
90 and over	8	—	—	—	—	—
Total	2576	55	49	70	102	276

TABLE III.

Gall-stones in Fourth, Fifth, Sixth and Seventh Thousand Autopsies (Female Subjects).

Age Group. (Years.)	Number of Autopsies.	Types of Gall-stones.				Total.
		Facetted.	Solitary.	Pigment.	Unspecified and Mixed.	
Under 10	1	—	—	—	—	—
10 to 19	54	1	—	—	—	1
20 to 29	145	1	1	—	1	3
30 to 39	163	6	5	5	3	19
40 to 49	215	11	9	4	8	32
50 to 59	291	20	18	12	21	71
60 to 69	310	27	20	13	25	85
70 to 79	179	17	19	14	14	64
80 to 89	48	9	4	—	5	18
90 and over	4	—	—	—	—	—
Age not stated	2	—	—	—	—	—
Total	1412	92	76	48	77	293

surprising that none of the 49 with pigment stones had either of these conditions.

Summary.

1. Gall-stones in Australia are twice as common in women as in men.

2. They begin to be common in men in the fifties (one in 11) and in women in the thirties (one in nine).

3. One at least in six men aged over seventy years, and one in each three women aged over 70 years will have gall-stones.

4. The pigment type seems the most common in males and the facetted in females.

5. There does not seem to be any association between gall-stones and malignant disease, or between gall-stones and cardiac infarction or fibrosis.

RECURRENT RECTAL PROLAPSE: AN OPERATIVE METHOD OF TREATMENT.

By KENNETH W. STARR,
Sydney.

THE revealing follow-up on patients from Saint Mark's Hospital (Hughes, 1949) suffering from rectal prolapse has disturbed proctologists. A recurrence rate of 60% after proctosigmoidectomy has instituted a search for an operation to replace this unsatisfactory and ofttimes bloody procedure. A suggested operation is here presented. The procedure was first mentioned in 1948, having been performed in 1947.

It is based upon two complementary concepts of the causal pathology of rectal prolapse: firstly, anal prolapse, which derives from a weak anal sphincteric mechanism; secondly, rectal prolapse, which is a sliding hernia of Douglas's pouch through the weak pelvic floor between the *levatores ani*. The operation is designed to reinforce the anal sphincter by using interlocking *fascia lata* slings (Cato) and to obliterate the pouch of Douglas (Roscoe Graham), and is followed by sigmoidopexy.

Three stages are pursued: (*a*) the patient is prone and a *fascia lata* graft is cut from the left ilio-tibial tract with a stripper; (*b*) the patient is in the lithotomy position and the *fascia lata* slings are inserted about the anus; (*c*) the patient is in Trendelenburg's position, and left iliac laparotomy is performed.

The details are as follows.

Pre-Operative Phase.

Anæmia and cough are alleviated. A week's preparation with phthalylsulphathiazole given by mouth and bowel lavage is followed by two days of intramuscular administration of penicillin and streptomycin before operation; this is continued for five days after operation.

Operative Phase.

Movement of the patient should be reduced to a minimum and if this is achieved blood transfusion is not required. As a precautionary measure, isotonic glucose infusion may be set up for use in case of urgency. As there is no blood loss with this method (unlike proctosigmoidectomy), and it represents an æsthetic surgical procedure, shock is the direct result of movement and operative time. Spinal anæsthesia and "Pentothal" supplement are preferred, but not essential. In lieu of the *fascia lata*, the plantaris tendon is admirable, and has been used on several occasions. It is readily found through a small medial calf incision, and removed by the use of the stripper through a medial ankle incision.

Two slings, each eight inches long and half an inch wide, are required. The patient is in the lithotomy position and two incisions are required for each sling. First, a two-inch oblique incision is made over the lower border of the *gluteus maximus*, at the level of the anus (Cato, 1935). A long Moynihan's cholecystectomy forceps is passed from this wound subcutaneously to the opposite side of the anus anterior and posterior to the orifice. A small incision (three-quarters of an inch) is made over the tip of the forceps and the ends of the *fascia lata* strip are caught in the forceps and withdrawn *seriatim*. A sling is thus formed about the anus, the ends protruding from the lateral wound. The sling is completed by performing the same operation on the opposite side. The slings are then drawn tight and attached to the gluteus fascia with interrupted chromicized catgut number 00, so that the anus admits the tip of the little finger. The wounds are closed with interrupted silk sutures.

The patient is then placed in the Trendelenburg position and a left iliac laparotomy is performed. There are two objects to be achieved at this stage—namely, obliteration of Douglas's pouch (the hernial sac which is lined by thickened peritoneum), and sigmoidopexy (Graham, 1935). The colon is first drawn well up into the left iliac fossa and a horseshoe incision (as in the mobilization of this section in abdomino-perineal resection) is made alongside the bowel, descending into Douglas's pouch and ascending along the opposite side of the gut. This enables the rectum to be drawn upwards and its anterior wall sewn by interrupted chromicized catgut sutures to the peritoneum on the anterior wall of Douglas's pouch. The posterior wall of the pouch is covering the rectum and is now at the level of the iliac fossa. This bowel segment is now fixed by multiple sutures to the peritoneum and muscle of the iliac fossa to ensure a firm sigmoidopexy. The iliac wound is closed in layers.

Post-Operative Management.

Post-operative management presents no real difficulties. Diarrhœa yielding to opium has been noted in one case, and this proved troublesome. Mucoid discharge has continued from the anus for one to three months after the operation, but caused no disablement.

The mental, social and economic status of those patients whose prolapse recurred after proctosigmoidectomy performed by competent and expert surgeons, may be worthy of emphasis. After the prolapse had been cured the outlook was so changed that the report of the cases and of the method was considered to be justified.

Reports of Cases.

CASE I.—H.B., a female patient, aged sixty-three years, developed a fist-sized rectal prolapse in 1946, and was treated by proctosigmoidectomy in February, 1946. Recurrence was fully developed by June, 1947, when the operation described was performed. From being a housebound, miserable woman, she now led her former active life. While moving a refrigerator in 1949, prior to a Christmas party, she felt a "snap" at her anus, and it was obvious on examination that one of her anal slings had broken. It was presumed that a new sling would be required, but in two weeks complete healing had occurred. During this period, paraffin by mouth and a sedative suppository after a sitz bath each day were prescribed. In March, 1950, prior to another social affair, she divided the left middle finger flexors in the palm on a bottle, and this injury required operation.

CASE II.—C.G., a female patient, aged sixty-one years, although a farmer's wife and very busy, suffered from ankylosing spondylitis of moderate degree. The advent of *prolapsus uteri* in 1945 was an added handicap to her, and perineorraphy was performed. A vaginal stricture subsequently developed. In 1946 a rectal prolapse (fist-sized) appeared and proctosigmoidectomy was advised and performed. Recurrence took place early in 1947, and for two subsequent years she had led a miserable life. Surgical repair as described was undertaken in November, 1949. Her post-operative convalescence was marred by diarrhœa from the third to the sixth day, for which aureomycin was used. Moniliasis of the perineum developed in the second post-operative week and presented some difficulty in cure (superficial X-ray therapy was eventually required). In March, 1952, Z plastic procedures were used for the cure of the vaginal stricture. She had subsequent deep X-ray therapy for her ankylosing spondylitis, and declares that she is a "new woman". The rectal prolapse has remained cured for three years.

CASE III.—H.K., a male patient, aged forty-three years, sustained a rectal prolapse in 1946 and was treated by proctosigmoidectomy in 1947. The recurrence took place in 1948 and was operated upon in August, 1948. Convalescence and subsequent follow-up require no comment. Cure has been achieved for four years.

Comment.

For mental and social cripples, perianal insertion of a wire remains the easiest and best surgical procedure; but active and responsible individuals require a better prospect. The procedure outlined appears worthy of trial.

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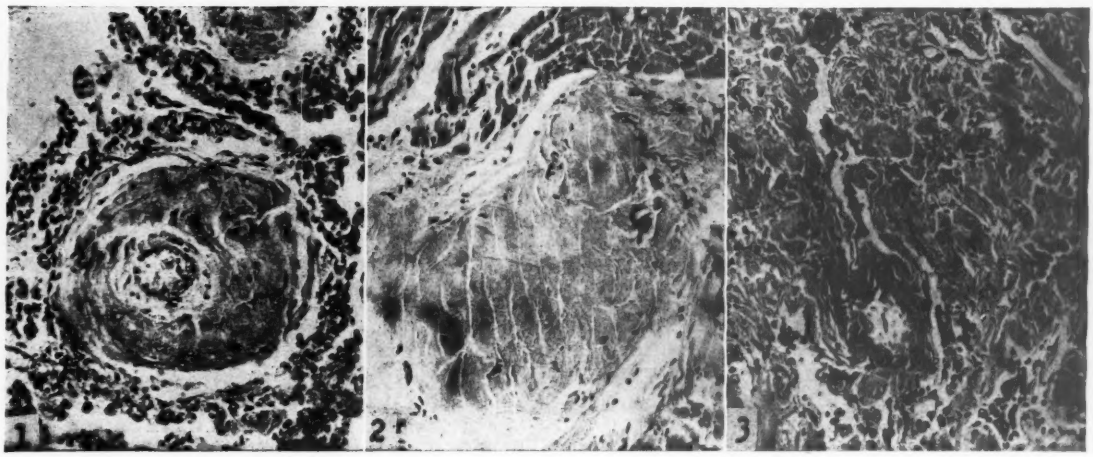
ILLUSTRATIONS TO THE ARTICLE BY A. K. COHEN.



ILLUSTRATIONS TO THE ARTICLE BY C. H. CAMPBELL, M.B., B.S.

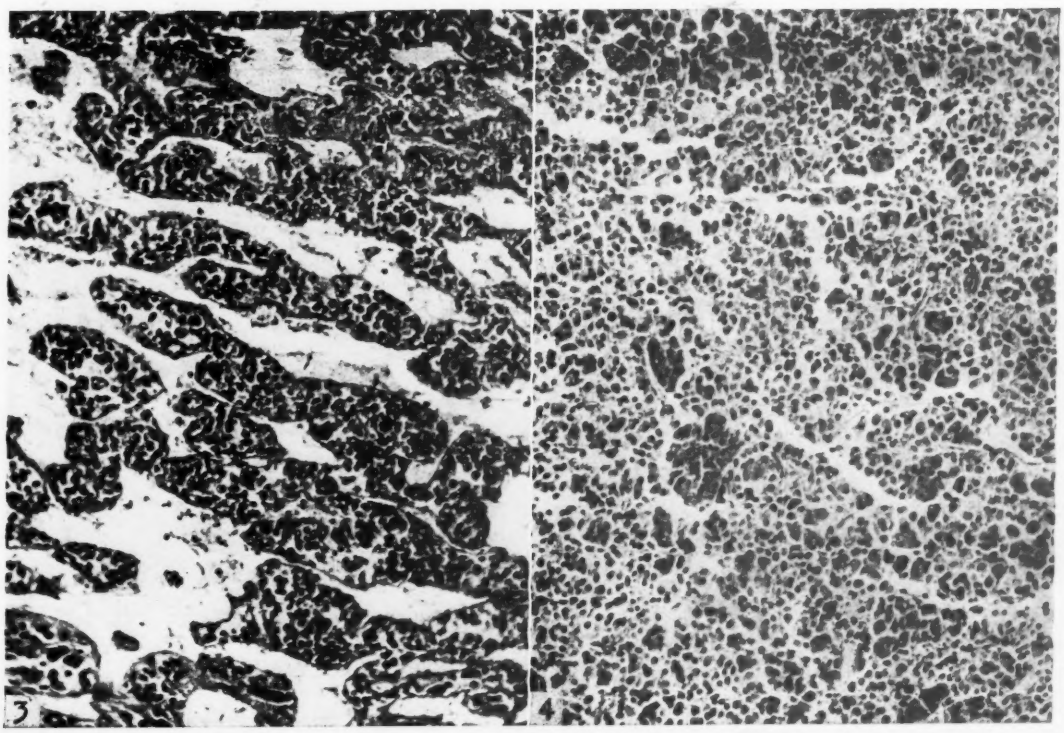


ILLUSTRATION TO THE ARTICLE BY RICHARD FLYNN AND LEO FLYNN.

FIGURE I.

ILLUSTRATION TO THE ARTICLE BY RICHARD FLYNN AND JOHN H. HALLIDAY.

FIGURE II.

ILLUSTRATIONS TO THE ARTICLE BY IAN F. POTTS, M.S., F.R.C.S.

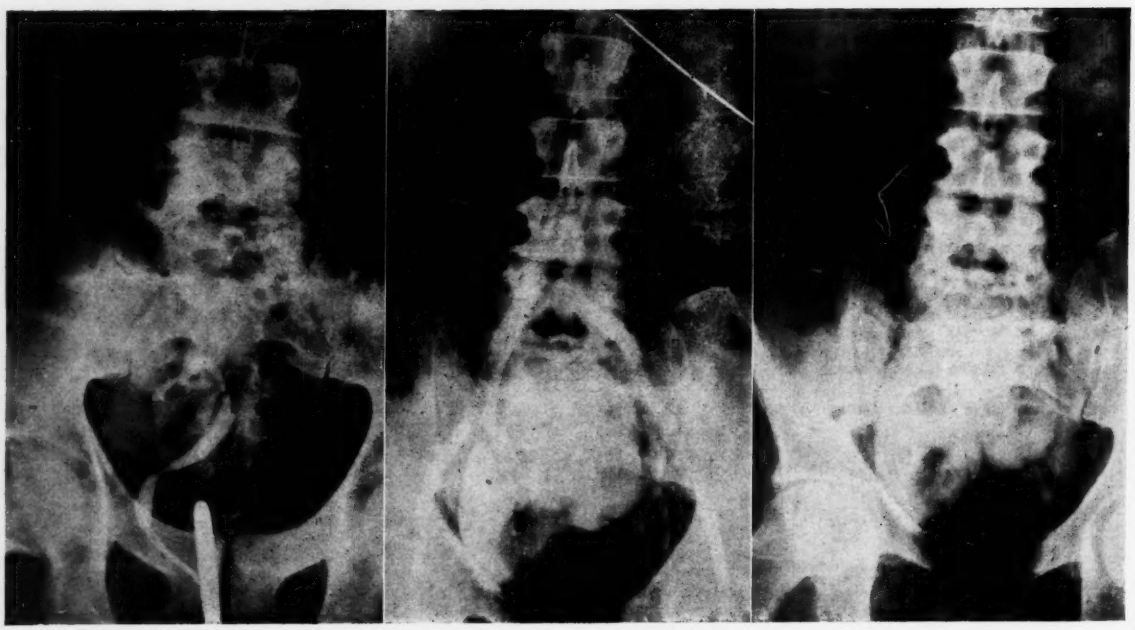


FIGURE I. FIGURE II. FIGURE III.

Reports of Cases.

PRIMARY SYSTEMIC AMYLOIDOSIS, WITH THE REPORT OF A CASE.

By A. K. COHEN,
Department of Pathology, University of Adelaide.

AMYLOID was first named by Virchow in 1853 because of its similarity to starch in reaction with acid-iodine. He recognized that it occurred in association with suppurative disease elsewhere in the body. Wild in 1886 reported the first case of primary systemic amyloidosis, and since that time some seventy cases of the primary disorder have been reported in the literature.

Amyloid occurs most commonly as a result of chronic inflammatory diseases. Such conditions include tuberculosis, syphilis, ulcerative colitis, bronchiectasis, rheumatoid arthritis and osteomyelitis; it may also occur as a sequel to thermal burns. In this secondary type of amyloidosis the organs mainly involved are the liver, spleen, kidneys and adrenals. The material stains in a characteristic manner with acid-iodine and congo red, and metachromatically with methyl violet; it is found extracellularly in the form of an infiltration between the endothelium of the capillaries and the parenchymal cell. Encroachment on the parenchyma may be so severe as to cause failure of the organ quite apart from the original disease, and uræmia, cholæmia and Addison's disease (Stemmerman and Auerbach, 1944) have all been reported as caused by amyloidosis. Recovery may occur after the removal or cure of the causative factor (Trassoff *et alii*, 1944); but the one-year mortality rate after the onset is approximately 80% (Pearlman, 1951).

Several other types of amyloidosis are recognized—notably in association with multiple myeloma, as localized tumour formations, and in the isolated skin disease *lichen amyloidosis*. Occasionally localized amyloid degeneration with varying staining properties may be found at the periphery of new growths and inflammatory lesions, and rarely as an incidental discovery in the heart valves and seminal vesicles of the aged (King, 1948).

Primary systemic amyloidosis differs from the other forms in several important respects. No preceding disease is found and there is a tendency to involve the heart, lungs, striped muscle, gastro-intestinal tract, tongue and skin; there is little or no involvement of the liver, spleen, kidneys and adrenals, which are affected by secondary amyloidosis. The lesions themselves are frequently nodular, and they take the recognized stains for amyloid poorly if at all. However, this distinction is not absolute, as in some cases there is preponderantly "secondary' distribution, in some there is localized tumour formation, and in others there may be involvement of only one system such as the cardio-vascular system. The nervous system, both central and peripheral, is almost always spared. The diagnosis of the condition is difficult, partly because of its rarity, but also because of the diffuse nature of the lesions and the absence of typical symptoms and signs. Nevertheless, there is an increasing volume of literature on the condition, and several reviews have appeared in the last ten years, from which emerges a recognizable clinical syndrome (Koletsky and Stecher, 1939; Lindsay and Knorp, 1945; Eisen, 1946; Goltz, 1952).

While the underlying nature of the chemistry and formation of amyloid remains obscure, classification of the disease must be tentative; perhaps all cases now regarded as primary will in time be regarded as secondary to some yet undiscovered cause. The classification suggested by Reimann, Koucky and Ecklund (1935) preserves the essential differences between the various forms of the disease: (i) primary systemic amyloidosis, (ii) secondary amyloidosis, (iii) localized or tumour-forming amyloidosis, (iv) amyloidosis secondary to multiple myeloma.

Report of a Case.

A widowed pensioner, aged seventy-one years, was admitted to the Royal Adelaide Hospital on January 29, 1953. She had been well until eighteen months before, when she began to suffer sharp pains in the fingers of both hands which were worse when she lay down. Low back pain had commenced in the last three months and was followed by pain down both legs of no definite distribution. She needed assistance to walk. For one month her tongue had become progressively more swollen and she experienced difficulty in talking and swallowing. There were now continual paræsthesiæ of the fingers. She had lost eight pounds in weight over the last month. Her previous health had been good and her exercise tolerance had been fair for her age. There was no history of tuberculosis.

On examination of the patient, the temperature was 98·6° F., the pulse rate was 120 per minute and the pulse was regular, and the respirations numbered 24 per minute. She was a thin, elderly, grey-haired woman with slurred speech. There was a pale, diffuse, waxy thickening of the skin, which extended from the skin of the lower lip over the anterior and posterior triangles of the neck and onto the anterior chest wall. Beneath the thickening in the neck numerous small mobile glands could be felt. The tongue was greatly enlarged, and filled the whole of the mouth; the surface was pale, but the normal papillæ were preserved and its mobility was not greatly impaired. The muscles of the thenar and hypothenar eminences were wasted, and the overlying skin had an erythematous appearance. The remainder of the skin of the hands was pale. There was ulnar deviation of the first and second fingers with radial deviation of the third and fourth. Flexion of all the interphalangeal joints, particularly the terminal joints, was limited. The other joints appeared normal. The reflexes were present and active and there were no sensory changes. The apex beat was in the fifth left intercostal space in the mid-clavicular line, and the heart sounds were clear and regular. The blood pressure was 155 millimetres of mercury, systolic, and 80 millimetres, diastolic. There were scattered rhonchi over both lungs. The liver was palpable two centimetres beneath the right costal margin. Apart from slight œdema of the ankles there were no other abnormalities.

The urine contained an occasional trace of albumin. The specific gravity varied between 1010 and 1024. No examination for Bence-Jones protein was made. The hæmoglobin value was 11·2 grammes *per centum* (73%). The red blood cells numbered 3,900,000 per cubic millimetre, the white blood cells numbered 3,400 per cubic millimetre, and the differential count gave normal results. X-ray pictures of the chest, hands, shoulders and skull were normal. The serum protein content was 5·7 grammes *per centum*, and the serum calcium content was 5·9 milliequivalents per litre. The fæcal occult blood test gave a faintly positive result. The Wassermann test produced a negative result. The skin condition was thought to be a localized form of scleroderma, and biopsy of the skin from the neck was reported as revealing atrophy of the covering squamous epithelium and increase of collagenous fibrous tissue in the subcutaneous layers.

The patient was encouraged to sit out of bed and physiotherapy was commenced. During the next fortnight there was some improvement in the movement of the joints, but swallowing became progressively more difficult until she could take only fluids. On February 22 she fell from bed and sustained an impacted fracture of the head of the right humerus, after which her condition deteriorated; she developed bronchopneumonia and died on March 6.

Autopsy was performed six hours after death. The skin lesions and wasting of the hands were as described. The pectoralis, sternomastoid and intercostal muscles were extremely pale and appeared to contain an excess of fibrous tissue. The subcutaneous, pericardial, omental and retroperitoneal fat was everywhere dry and lobulated. There was no pleural effusion or ascites. The tongue was large and pale and on being cut had a waxy, greasy appearance. The pharynx, œsophagus, stomach and small intestine were involved in a curious thickening which was confined to their serous coats; they were pale, swollen and dry. At the pylorus and extending for 10 centimetres along the duodenum the thickening was granular and somewhat pink in colour, giving a cobbled appearance to the serous coat. The mucosa was normal on macroscopic examination. Examination of the remainder of the small and large bowel revealed varying degrees of diffuse involvement. The lungs were dark crimson in colour, and both lower lobes were affected by patchy lobular bronchopneumonia; they had a remarkable rubbery feeling and cut with some difficulty. Apical emphysema was present. The heart was small, the valves appeared normal and the coronary orifices were patent; the heart muscle itself was pale and appeared mildly fibrotic. The liver, spleen,

kidneys and adrenals were of normal size, and in them acute congestion only was seen. The brain and spinal cord appeared normal. There was no other significant finding.

Histopathological Findings.

Sections were stained with hæmotoxylin and eosin, Congo red and methyl violet. Unless the contrary is stated, the following description applies to the hæmatoxylin and eosin preparation.

In all the striped muscle taken for the preparation of sections (*pectoralis major*, sternomastoids, intercostals and rhomboids) were found separation of the muscle bundles and thickening of the sarcolemmal sheaths by a homogenous material which stained uniformly pink with eosin. Varying degrees of compression up to complete atrophy were seen, with loss of striation and shrinking of the muscle bundles. Examination of the blood vessels revealed diffuse thickening of the media, which at times encroached on the lumen of the vessel (see Figure I). Although the substance in the media of the vessels and that between the muscle fibres appeared the same on microscopic examination, only the former took up the Congo red stain, and neither was stained by methyl violet. The heart muscle showed similar changes, but here a nodular tendency was more pronounced, and in places a long strand of fibrous tissue which terminated in a nodule of amyloid could be traced from the epicardium into the depths of the muscle (see Figure II). This also failed to take the recognized stains. The smaller coronary vessels were likewise affected, although in no place was the lumen of the vessel seriously occluded by the medial infiltration.

In the lower lobes of the lungs widespread amyloidosis was present. The larger pulmonary vessels were heavily involved, with total occlusion of their lumen in many places. Interalveolar deposits of amyloid were seen, and these in conjunction with the vascular deposits gave the impression of a patchy amyloid "consolidation" (see Figure III). The bronchial mucosa, the muscle and the lymph glands were unaffected. There was an overlying bronchopneumonia. Vascular involvement alone was observed in the upper lobes of the lung. All lung amyloid stained brightly with Congo red.

The muscular fibres of the tongue were swollen and homogeneous, and the muscle bundles were separated by strands of amyloid material. Thickening of the muscle basement membrane and much interstitial œdema were present, but inflammatory cells were not observed. The œsophagus, stomach and small intestine were diffusely involved. The serosa and muscular layers were mainly affected, and examination of the muscle revealed localized swelling and the amyloid infiltration with surrounding thin fibrous tissue. Small vessels of the muscularis contained deposits of amyloid, but the mucosa was normal. The cobbled appearance of the serosa was due to clumps of infiltrated muscle surrounded by a thin shell of newly formed fibrous tissue. The liver was acutely congested with early centrilobular degeneration of the parenchymal cells and some interstitial œdema; the larger radicles of the portal vein and hepatic artery stained lightly with Congo red. The kidney was acutely congested, but only two large arcuate arteries were seen to contain amyloid. One section of the ulnar nerve contained amyloid in the media of the *vasa nervorum*, but the axons and myelin sheaths appeared normal. Sections of the spinal cord at various levels were normal.

The final pathological diagnosis was primary systemic amyloidosis involving the gastro-intestinal tract, tongue, lungs, heart and striped muscle, with terminal bronchopneumonia.

Comment.

While the precise composition of amyloid is uncertain, it is known to consist mostly of protein and to contain a small quantity of polysaccharide. Hass and his co-workers (Hass, 1945) have shown that the polysaccharide is identical with or closely related to chondroitin sulphuric acid. They have detected differences in the composition of the amyloid produced experimentally in different species of animals and between the amyloid of tuberculosis and that secondary to multiple myeloma in man (Hass *et alii*, 1943). This experimental evidence, together with the observed variation in distribution of the human lesions and the unpredictable staining reactions, suggests that there is more than one form of amyloid. Hence the material recognized histologically as amyloid probably has a variable chemical composition and comprises a group of closely related substances.

The experimental production of amyloid is usually associated with a rise in serum globulin content. This fact, together with the knowledge that hyperglobulinæmia frequently accompanies the human disease, suggested that the amyloid deposits are the result of an overwhelming deposition of globulin in the tissues (Eklund and Reimann, 1936). However, in the reported cases of primary systemic amyloidosis, the occurrence of a raised serum protein level is very rare, and in the case here reported the protein level was normal. This objection is countered by the observation that in the experimental animal the serum globulin level begins to fall as amyloid is deposited. Magnus Levy (1931) drew attention to the frequent association of multiple myeloma with amyloidosis. Bayrd and Bennett (1952) state that the incidence of amyloidosis in multiple myeloma is approximately 15%. Amyloid and Bence-Jones protein are chemically closely related, as is seen by the affinity of both of them for Congo red; but there is no evidence that one is converted to the other. The amyloid occurring in association with multiple myeloma has a similar distribution to that of the primary form, and Apitz (1940) believes that all cases of primary amyloidosis are due to a myeloma not found at autopsy. Bayrd and Bennett (1952) report a case in which differential staining reactions suggested that amyloid was present within the myeloma cells themselves; since myeloma cells are not known to be phagocytic, they hold that this finding indicated that the amyloid was formed within the cells. Trubowitz (1950) noted that amyloid was present in the plasma cells and polymorphonuclear cells of the bone marrow aspirated from a patient with multiple myeloma, and suggests that the cells may have become phagocytic in much the same way as the L.E. cell of Hargrave (Hargrave *et alii*, 1948). Whether plasma cells form amyloid or whether it is formed in the bone marrow is still unsettled. Koletsky and Stecher (1939) observe that many of the primary cases reported in the literature have not excluded multiple myeloma by the employment of specific diagnostic techniques. In the present case the normal X-ray findings, the normal serum protein content and the absence of autopsy evidence make this diagnosis extremely unlikely.

From the collected cases of primary systemic amyloidosis a recognizable clinical picture has appeared. The average age incidence is fifty-six years, but the range is between thirty-four and ninety years (Eisen, 1946). Most of the cases occur between the sixth and eighth decades. The sexes are equally affected and there appears to be no occupational predisposition. The mean survival after the onset of symptoms is thirty-two months (Eisen, 1946); but Koletsky and Stecher (1939) report a case in which symptoms lasted fourteen years. Whilst regression of secondary amyloidosis has been reported, in no case of primary amyloidosis has this apparently occurred.

Cardio-vascular System.

The cardio-vascular system is involved in over half the cases (Lindsay, 1946). Patients commonly present with congestive cardiac failure of obscure ætiology, but since they are in the age group in which heart disease is common, there is a tendency to ascribe their symptoms to atherosclerotic or hypertensive cardiac failure. Hypertension is not a feature of the disease, and the few patients who are hypertensive show a progressive fall in blood pressure with the worsening of their disease (Eisen, 1946). Lindsay (1946) states that amyloidosis may produce cardiac failure in a number of ways: (*a*) amyloidosis of the pulmonary vessels inducing chronic *cor pulmonale*, (*b*) deposition in the blood vessels of the heart, (*c*) diffuse or nodular infiltration of heart muscle with compression of muscle bundles, (*d*) pericardial or endocardial deposits, (*e*) extensive valvular deposits producing stenosis or insufficiency.

Macroglossia.

Macroglossia is one of the most helpful diagnostic signs, although it is not an invariable accompaniment of the condition. The enlargement is usually uniform, and tenderness is absent, although ulceration may occur because of

the increased size. There may be difficulty in articulation or swallowing, as in the present case, or the amyloid may infiltrate the whole floor of the mouth, the cheeks and the submandibular region, leading to diffuse induration. Enlargement of the tongue may occur rapidly in the course of days or weeks, or may be more gradual, taking up to two years for its appearance. The surface of the tongue is usually pale and dry, the body diffusely enlarged and firm, and the mobility of the organ may be impaired by the size and texture. Death may be hastened by the obstructive effects of this organ.

Gastro-intestinal Tract.

Symptoms referable to the gastro-intestinal tract occur in about 10% of cases (Eisen, 1946), although actual involvement of the intestines is much more common. Unlike secondary amyloidosis, in which deposits are found in the mucous membrane, in primary amyloidosis the deposits occur in the smooth muscle of the muscularis, the muscularis mucosa and the blood vessels of the submucosa. There may be abdominal pain, intestinal cramps or even hæmatemesis as in Golden's case (Golden, 1945). Jaundice is extremely rare (Orloff and Felder, 1946). In the case reported here there were no symptoms referable to the gastro-intestinal tract despite the severe involvement.

Skin and Mucous Membrane.

Goltz (1952) in a recent review states that the most common cutaneous manifestation of the condition is the presence of hæmorrhages into the skin. Purpura may occur in normal skin or in skin which shows other signs of amyloid infiltration, such as diffuse thickening or pale papular lesions. It may be the presenting symptom. Some authors ascribe the bleeding tendency to amyloid infiltration of the small cutaneous vessels; but perivascular hæmorrhage in other sites of the body is rare in this condition. No consistent abnormality of the clotting mechanism has been demonstrated. The diffuse thickening of the skin resembling scleroderma, which was noted in the present case, is also relatively common and may involve the head and neck, the hands and the ano-genital area. Other cutaneous manifestations include translucent papules, nodules, tumours and plaques (Goltz, 1952).

Skeletal Muscle.

Infiltration of skeletal muscle with accompanying weakness occurs in nearly half of the reported cases. However, wasting is not the rule, and the muscles are firm and somewhat enlarged (Eisen, 1946). Amyloid is deposited around the muscle fibres and less frequently within the sarcolemmal sheaths. There are atrophy and degeneration of the muscle, presumably due to pressure.

Peripheral Nervous Involvement.

Peripheral nervous involvement is extremely uncommon (Kernohan and Woltman, 1942), and the changes seen in the *vasa nervorum* in this case were not accompanied by clinical or pathological involvement of the nerves. The brain and spinal cord are usually spared.

Pulmonary Amyloid Deposits.

Pulmonary amyloid deposits are a common autopsy finding, and present a similar appearance to that described in this case. The severity of the lesions varies and may be so extreme as to cause hypertrophy of the right ventricle and ultimate heart failure apart from involvement of the heart itself. Radiological evidence of pulmonary amyloidosis is rare, even when, as in this case, the infiltration is massive and widespread.

The liver, spleen, kidneys and thyroid may show amyloid deposition, but only in a few cases has this been severe enough to interfere with function (Dahlin, 1949).

Primary systemic amyloidosis may be suspected clinically in any case of congestive cardiac failure in which the ætiological background is obscure, and especially in any case in which lesions of the skin, tongue, mucous membranes and skeletal muscle of a diffuse and hypertrophic nature are present. Biopsy is the only reliable means for securing a definite diagnosis, and the specimen may be taken from skin or the tongue or by needle aspiration of the liver. The condition may be overlooked unless the unusual staining characteristics of the material are realized and a number of reagents employed. The Congo red retention test will be helpful only in those cases in which the amyloid takes the stain, and in the presence of cutaneous amyloidosis may leave a permanent coloration of the skin. The presence of multiple myeloma should be excluded in all cases in which the diagnosis is made.

Summary.

The commoner clinical and pathological features of primary systemic amyloidosis are discussed. A case is presented which conforms with previous descriptions of the disease. This was a case of primary systemic amyloidosis occurring in a woman, aged seventy-one years. Dysphagia and dysarthria due to macroglossia, progressive muscular weakness with wasting of the thenar and hypothenar muscles, and a diffuse thickening of the skin of the neck and the anterior chest wall were the main features.

There were amyloidosis of the skin, tongue, alimentary tract, lungs, heart and striped muscle, as well as deposition of amyloid in the media of most of the small blood vessels. The staining reactions of the amyloid were variable. Despite the considerable infiltration of the stomach, duodenum and intestines and the widespread involvement of both lungs, there were no symptoms directly referable to these organs. X-ray examination of the chest failed to reveal the pulmonary amyloidosis. Amyloid was found in the *vasa nervorum* of the peripheral nerves, but no degeneration of the nerves themselves was present. The cause of death was bronchopneumonia, to which the difficulty in swallowing contributed.

Acknowledgement.

I am grateful to Dr. K. S. Hetzel, honorary physician at the Royal Adelaide Hospital, for permission to discuss the case presented in this paper.

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Legends to Illustrations.

FIGURE I.—Medial infiltration of a pulmonary vessel with narrowing of the lumen

FIGURE II.—A nodular amyloid deposit within the myocardium.

FIGURE III.—Diffuse amyloidosis of the lung.

FUNCTIONAL ADRENAL CORTICAL CARCINOMA COMPLICATED BY A PERFORATED GASTRIC ULCER: REPORT OF A CASE WITH POST-MORTEM FINDINGS.

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IT has become apparent that peptic ulceration is one of the complications which may arise during treatment with some of the adrenal gluco-corticoid hormones or ACTH. It is of some interest, therefore, to report a case of an adrenal cortical carcinoma which was complicated by a peptic ulcer, particularly as there was evidence of the production of a gluco-corticoid-like hormone by the tumour.

Kepler *et alii* (1948) have reported a case in which these two conditions were present, but they were not thought to be related.

Clinical Record.

E.A., a female patient, aged seventy-two years, was admitted to the Eye Department of Sydney Hospital on August 14, 1952, with a cataract in the left eye. On account of her general condition she was discharged and referred to the general outdoor department for investigation. On August 23 she was admitted to the gynæcological ward with a provisional diagnosis of carcinoma of the body of the uterus, *diabetes mellitus* and hypertensive congestive cardiac failure.

When questioned about her illness, she said that she had "no complaints"; further interrogation brought the following symptoms to light: vaginal bleeding of three months' duration (necessitating the use of three pads per day), increasing breathlessness on exertion for ten years, so that now she became breathless on walking a few steps, swelling of the ankles of one month's duration, and "an indigestion pain" of doubtful duration. Her menstrual history had been normal up to the menopause at the age of forty-six years. There had been no vaginal bleeding since then until this illness. She had had one miscarriage and eleven children, all born normally.

The patient was a short, obese, plethoric woman. Her pulse rate was 90 per minute and the pulse was regular in time and amplitude. The heart was enlarged on clinical examination. The heart sounds were normal. The blood pressure was 160 millimetres of mercury, systolic, and 110 millimetres, diastolic. There was gross pitting œdema of the whole of the lower limbs. The respirations numbered 22 per minute. The percussion note was dull and there were crepitations at the bases of both lungs. The abdomen was swollen and obese, and there was doubtful ascites. The external genitals were normal. On vaginal examination, the cervix had a ragged nobbly anterior lip, the uterus was retroverted and slightly enlarged. The specific gravity of the urine was 1018. There was a "+" reaction for albumin, "+++" for sugar and "++" for acetone.

A number of investigations were carried out, with the following results. The hæmoglobin value was 14·2 grammes *per centum*; the blood urea nitrogen content was 12 milligrammes per 100 millilitres of blood; the blood creatinine content was 1·5 milligrammes per 100 millilitres of blood; the standard urea clearance was 78%; the blood sugar content was 129 milligrammes per 100 millilitres in the fasting state, 195 milligrammes per 100 millilitres half an hour after the ingestion of 50 grammes of glucose, and 221 milligrammes per 100 millilitres two hours after the ingestion of 50 grammes of glucose. X-ray examination of the chest showed an increase in the transverse diameter of the heart and congestive changes in the lung fields. The urinary 17-ketosteroid estimation was 11·8 milligrammes per twenty-four hours specimen.

The patient was subsequently transferred to the care of a physician and received a 1700 Calorie salt-free diabetic diet, "Digoxin" (0·25 milligramme) and aminophylline (100 milligrammes) three times a day, injections of ordinary insulin, 15 units at 7.30 a.m. and 12 noon, and 10 units at 4.30 p.m., and intramuscular injections of "Thiomerin", two mils per day, twice a week. The glycosuria was controlled up till a few days before her death. There was no response to the mercurial diuretic. No alteration in her clinical condition occurred until September 12, when suddenly at 6 a.m. the patient had a severe epigastric pain. When she was examined the pulse rate was 80 per minute, and the pulse was regular in time and amplitude. The heart sounds were normal. The blood pressure was 150 millimetres of mercury, systolic, and 80 millimetres, diastolic. There was some tenderness in the epigastrium, but no guarding. The patient was afebrile and there was slight glycosuria. At 8 a.m. the pain was still severe. She vomited five ounces of thick, viscid, bile-stained fluid. Her abdomen now was distended and resonant, and the blood pressure had dropped to 90 millimetres of mercury, systolic, and 70 millimetres, diastolic. A provisional diagnosis of acute myocardial infarction was made. Later, hæmorrhage began *per vaginam*, and she became very dyspnœic and cyanosed and died at 5.10 p.m. the same day.

After the autopsy findings were known, the following history was obtained from the patient's daughter:

> It all happened so very quickly. Mother was quite well up till April, 1952. She complained of feeling tired, so I took her along to the local doctor. He found that she had blood pressure. [The local practitioner states that the patient had suffered from blood pressure for some years.] In May mother noticed that she had difficulty in getting up stairs. I noticed that her face started to swell and became very red. Her features broadened and I took her to the doctor again. He found that she had diabetes. Mother's ankles then began to swell and she bled "internally". The hair on my mother's arms and, over the last two months, the hair on her face, grew very thick.

When questioned, the daughter said that her mother had always had a slight growth of facial hair, but it had increased greatly with this illness. She thought that she had first noticed the increased growth of the hair on the arms twelve months earlier. Her mother had gained a lot of weight. There was no family history of hypertension or diabetes.

Post-Mortem Report.

The autopsy was performed thirteen hours after death. The body weight was 73·5 kilograms (162 pounds) and the height 154·9 centimetres (five feet one inch).

The body was that of a short, elderly, obese female. The skin of the face, shoulders and neck was a reddish-purple colour. There was a heavy growth of short-cropped hair on the upper lip, the chin and the sides of the face, and of long hair on the forearms. The abdomen was extremely distended and tympanitic. There were several

non-pigmented striæ present on the lower abdominal wall. The lower limbs were grossly swollen and pitted deeply on pressure. There was a slight pitting œdema of the dorsum of the hands.

The thyroid gland was of normal size and the right lobe contained a small white nodule measuring 0·8 centimetre in diameter. The right pleural cavity contained 284 cubic centimetres of a straw-coloured effusion. Both lungs were intensely congested. A few small isolated yellow nodules were scattered throughout the lung tissue. The heart (340 grammes) was slightly enlarged, mainly owing to hypertrophy of the wall of the left ventricle, which measured 1·8 centimetres at the anterior papiliary muscle. The mitral ring was slightly dilated. The coronary arteries and the aorta were affected by a slight degree of atheroma.

A large amount of free air escaped when the peritoneal cavity was opened; it also contained 2·27 litres of a turbid, reddish-brown fluid. The peritoneum of the posterior abdominal wall, of parts of the lesser omentum and of the small bowel, was slightly congested. Numerous soft, yellowish deposits of newgrowth were scattered throughout the peritoneal cavity.

There was a large oval ulcer (3·5 by 3·0 centimetres), which had perforated, on the posterior wall of the body of the stomach (Figure I). There were no adhesions between the stomach and the peritoneum of the posterior wall of the lesser sac. There were a few small, isolated deposits of newgrowth in the liver. The spleen and the pancreas appeared normal.

FIGURE I.

The stomach opened along the greater curvature; an ulcer with a large perforation is present on the posterior wall.

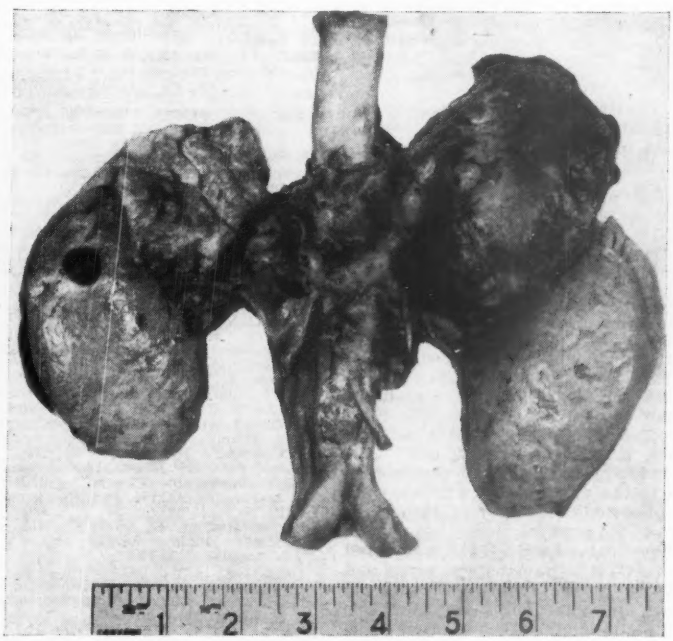

FIGURE II.

The left adrenal tumour is shown resting on the left kidney. The left adrenal and left renal vein are distended with tumour tissue. The right adrenal gland is small and atrophic.

The right adrenal gland was small but otherwise normal. The left adrenal gland (Figure II) was completely replaced by a soft, yellowish, encapsulated, bun-shaped tumour measuring 8·5 by 7·0 by 5·0 centimetres. The cut surface was yellow and there were numerous areas of hæmorrhage and necrosis. A large vein passed from the medial border of the tumour into the left renal vein. The left adrenal vein and the left renal vein were completely filled with tumour tissue, which extended into the inferior *vena cava* for a distance of 2·0 centimetres.

The kidneys were of normal size. Their capsular surfaces were finely granular and the cortices were slightly narrowed. The left kidney was not infiltrated by the adrenal tumour.

The uterus was uniformly enlarged and measured 10·0 by 5·0 by 4·0 centimetres. The endometrium was slightly hæmorrhagic. The vaginal surface of the cervix was irregular and covered with a thick, brown, mucoid exudate. Numerous small cysts containing a brown mucoid secretion were present in the cervix. The ovaries, bladder and rectum appeared to be normal.

The pituitary gland, which was damaged while being removed from the body, and the brain appeared to be normal. The bones were not examined. There was X-ray evidence of secondary deposits in the ribs.

Histological Findings.

Dr. R. Finlay-Jones reported in the following terms on the sections.

Examination of the left adrenal tumour showed the growth to be a cortical carcinoma. The pattern of the growth in some regions resembled that of the normal gland, the cells being arranged in anastomosing cords, separated by blood-filled sinuses (Figure III). In the tumour cells a moderate degree of pleomorphism was apparent (Figure IV). They were mainly round or polyhedral with abundant eosinophilic cytoplasm and eccentric hyperchromatic nuclei. Large multinucleated cells were fairly numerous. A special stain (Broster-Vines) demonstrated fuchsinophile granules in many cells.

The perforation in the stomach was lined with necrotic tissue. Fibroblastic proliferation in the adjacent stomach wall and infiltration with lymphocytes and plasma cells

were apparent. The absence of scar tissue indicated acute ulceration.

The sections of the pituitary gland were not satisfactory. Crook's hyaline change in the basophile cells was not present. There were secondary carcinomatous deposits in the liver, lungs, thyroid and omentum. There was a slight degree of venous congestion in the liver and lungs. The endometrium was of a non-secretory type. A slight degree of adenomyosis was present. In the kidneys there was a mild degree of nephrosclerosis, with slight hypertensive changes in the arterioles.

Summary of Post-Mortem Findings.

The post-mortem findings could be summarized as follows: (i) acute peritonitis, a sequel to the perforation of an acute gastric ulcer; (ii) a primary adrenal cortical carcinoma with metastases in the peritoneum, omentum, liver, lungs and thyroid; (iii) hypertensive congestive cardiac failure; (iv) adenomyosis; (v) chronic cystic cervicitis.

Discussion.

Kenyon (1944) has described six possible syndromes which may be associated with adrenal cortical carcinoma: (*a*) the adreno-genital syndrome; (*b*) Cushing's syndrome; (*c*) mixed clinical pictures, including features of the first two; (*d*) single or isolated endocrine disturbances—for example, diabetes; (*e*) feminizing syndromes; (*f*) tumours without endocrine manifestations.

In this case the plethora, the rounding of the facies, the hirsutism, the obesity, the diabetes and the value for the urinary 17-ketosteroid excretion (which was slightly raised for a woman of her age—Dorfman and Shipley, 1950), and possibly the œdema, together may be taken as evidence of hormone production by the adrenal tumour. Some of the features of Cushing's syndrome (Cushing, 1932) are present, but the case may best be regarded as an example of Kenyon's third group, the mixed clinical pictures.

The gastric ulcer was relatively acute, and apparently developed during the terminal illness. The following evidence suggests that there may be a relationship between it and the adrenal carcinoma.

1. The manifestations of Cushing's syndrome are thought to be principally due to an excess of adrenal gluco-corticoid hormones (Albright, 1942-1943). Overdosage with cortisone (a gluco-corticoid) has reproduced many of the features of Cushing's syndrome in some subjects (Williams, 1951).

2. The administration of ACTH and cortisone is associated with a great increase in the concentration of gastric pepsin and acid. The effect of ACTH presumably is due to an increase in the secretion of adrenal gluco-corticoids. Spiro, Reifenstein and Gray (1950) found that the administration of ACTH increased the excretion of uropepsin in normal subjects and in subjects with peptic ulcers, but did not increase the low uropepsin excretion found in patients with adrenal insufficiency, although cortisone did. Uropepsin represents the endocrine fraction of pepsinogen, and its rate of excretion in the urine varies with the rate of excretion of gastric pepsin (Janowitz and Hollander, 1951). These effects of ACTH and cortisone on gastric secretion were confirmed by Gray *et alii* (1951) who found that the administration of ACTH to humans was associated with a pronounced increase in the basal and nocturnal secretion of acid and pepsin approximating 200%. Cortisone was capable of producing a similar though less pronounced response. The maximum acid and pepsin effects were observed after seven to fourteen days of continuous corticotrophin therapy.

3. The effect of these hormones on gastric secretion is associated with the development of peptic ulcer in a significant percentage of subjects receiving the hormones, estimated by Halstead (1952) to be 4%.

Beck *et alii* (1950) first reported the occurrence of a perforated ulcer in association with ACTH therapy. Since then numerous cases have been reported of the occurrence, during or immediately after courses of ACTH or cortisone therapy, of a recrudescence or exacerbation of ulcer symptoms, of hæmorrhage from or perforation of a chronic peptic ulcer and of the development of peptic ulcer *de novo* which sometimes perforates (Gray *et alii*, 1951; Halsted, 1952; Habif *et alii*, 1950; Sandweiss *et alii*, 1950; Smyth, 1951; Lubin *et alii*, 1951; Kirsner *et alii*, 1952).

It is reasonable to infer that there was in this case, and that possibly in all cases of Cushing's syndrome, whatever the primary ætiology, an increase in the secretion of gastric pepsin and acid, which may lead to the development of a peptic ulcer in a susceptible subject.

Summary.

1. The case is reported of an elderly woman with an adrenal cortical carcinoma producing some of the features of Cushing's syndrome, who died from a perforated peptic ulcer.

2. Evidence is given that the administration of adrenal gluco-corticoid hormones is capable of producing some of the features of Cushing's syndrome and peptic ulcers in susceptible individuals.

3. The relationship between the ulcer and the adrenal cortical carcinoma is discussed.

Acknowledgements.

I wish to thank Dr. E. H. Stokes, senior physician at Sydney Hospital, for permission to publish this case. I am also indebted to Dr. H. M. Whyte and Dr. W. Simmonds for criticisms and suggestions in the preparation of this paper, and to Mr. R. Money for the illustrations.

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Legends to Illustrations.

FIGURE III.—Section of the adrenal tumour, showing a slight resemblance to the normal adrenal architecture.

FIGURE IV.—Section of the adrenal tumour illustrating the pleomorphism of the cells.

RUPTURED HYDATID CYST OF THE LIVER, WITH A FISTULA BETWEEN THE HEPATIC DUCT AND THE CYST.

By RICHARD FLYNN and LEO FLYNN,
Sydney.

A.B., a male patient, aged fifty-two years, sought medical attention on March 7, 1951, because of jaundice of three months' duration and on account of pain across the upper part of his abdomen. He stated that he had been well until December, 1950. At that time he had noticed a feeling of fullness in the epigastrium and a dull aching pain across the upper part of the abdomen. Two days after the onset of the pain he became deeply jaundiced. This jaundice had gradually cleared up until two weeks previously, when it suddenly returned. He had experienced no loss of appetite and no nausea, but had vomited twice. He still experienced a dull pain in his epigastrium, and since the onset of his trouble he had lost about one stone in weight. His stools were light in colour and his urine was dark.

On examination of the patient, apart from tenderness in his epigastrium on palpation and jaundice, physical examination gave entirely negative results. The following investigations were carried out. On March 29 the prothrombin index was 77. The serum alkaline phosphatase content was 19 units. The red blood cells numbered 4,300,000 per cubic millimetre, the hæmoglobin value was 12 grammes *per centum* and the white blood cells numbered 8200 per cubic millimetre.

Further liver function tests were carried out, with the following results. The thymol turbidity was one unit (normal range, 0 to 4 units). The Van den Bergh test produced a strong immediate direct positive reaction. The serum bilirubin content was 4·4 milligrammes per 100 millilitres. The serum alkaline phosphatase content was 28 units by King's method (normal range, 3 to 13 units). The total protein content was 6·8 grammes per 100 millilitres of serum; the albumin content was 4·8 milligrammes per 100 millilitres and the globulin content 2·3 milligrammes per 100 millilitres.

The prothrombin index on April 19 was 89. The patient had been receiving vitamin *K* injections since his admission to hospital.

A plain X-ray film of the upper part of the abdomen was suggestive of hydatid cyst, showing a small area of calcification. However, a Casoni test produced a positive reaction. After consultation we decided that laparotomy was indicated, so on April 23, through a right upper paramedian incision, exploration of the liver was performed. A cyst was found in the posterior part of the lower surface of the right lobe. The cyst was aspirated, but no fluid was withdrawn, the cyst having apparently drained its fluid contents into the duct. The cyst wall was then cautiously incised, and the endocyst and many daughter cysts were removed. The common duct was isolated and incised. Sounds were passed without difficulty into the duodenum, and a T tube was placed in the common duct, which was closed in the usual way around it. The long arm of the T tube and another drainage tube were pulled out through a stab wound in the usual way. The patient's convalescence was uneventful.

On May 15 an injection of "Abrodil" was made through the T tube and the choledochogram (Figure I) showed well the comparatively small dilatation of the common duct and the ruptured hydatid cyst opening into the duct of the right lobe. It also showed that there was no obstruction to the bile entering the duodenum. Vitamin *K* injections were continued and glucose was given intravenously for forty-eight hours. His convalescence was uneventful, the T tube was removed six weeks after operation, and no bile drained after that date. He has remained well since.

Comment.

The case is reported because, though rupture of a hydatid cyst of the liver into the bile ducts is relatively common, demonstration of the site of rupture is rare.

ADENOMA OF THE ADRENAL GLAND AND CARCINOMA OF THE BREAST.

By RICHARD FLYNN and JOHN H. HALLIDAY,
Sydney.

MISS E.M. was first examined in 1935 by one of us (J.H.H.). She was at that time aged fifty-one years, and stated that since the cessation of her menstrual periods four years previously she had become increasingly bald, but that growth of hair elsewhere on her body had greatly increased. Some years previously she had been informed that her uterus was of the infantile type.

On examination she was observed to have lost most of the hair on the vertex of the scalp (Figure I), the remainder being coarse and scraggly, with considerable

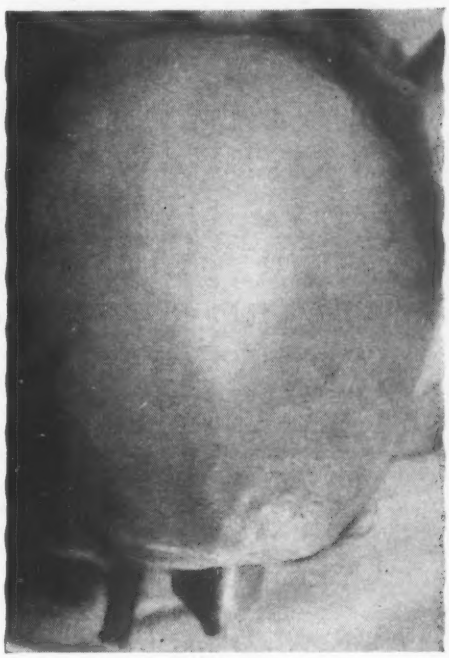

FIGURE I.

side-lever and moustache growth on the face. The pubic hair was of the masculine type of distribution, and the extremities and chest also exhibited considerable hirsuties. The voice was deep and gruff, and moderate hypertension was present.

No other significant findings resulted from examination of the blood, urine and skull (by X rays), and a tentative diagnosis of Cushing's syndrome was made.

She was next examined in 1947, when she gave a history of two recent attacks of renal colic, after the second of which she had passed two calculi. Her medical attendant had also observed a mass in the right breast. She presented much the same picture with regard to the hirsuties and the loss of scalp hair. The hypertension was now more severe (blood pressure 240 millimetres of mercury, systolic, and 130 millimetres, diastolic) and albumin was present in the urine. A hard, painless mass was present in the right breast.

After her admission to hospital the fasting blood urea content was found to be 52 milligrammes *per centum*. There was poor urea clearance, and an excretion pyelogram revealed multiple calculi and a large shadow above the

upper pole of the right kidney, which appeared to be displaced downwards (Figure II). The basal metabolic rate was estimated to be −17%, and the twenty-four hour urinary excretion of 17-ketosteroids was found to be 660 milligrammes.

Radical mastectomy was performed (R.F.) and the pathological report confirmed the diagnosis of adenocarcinoma of the breast with invasion of the axillary lymph glands.

A month later a large tumour of the right adrenal gland was removed through a subcostal incision. After a stormy post-operative period the patient made a good recovery.

Dr. G. Davies, pathologist of Royal Prince Alfred Hospital, reported on the tumour in the following terms:

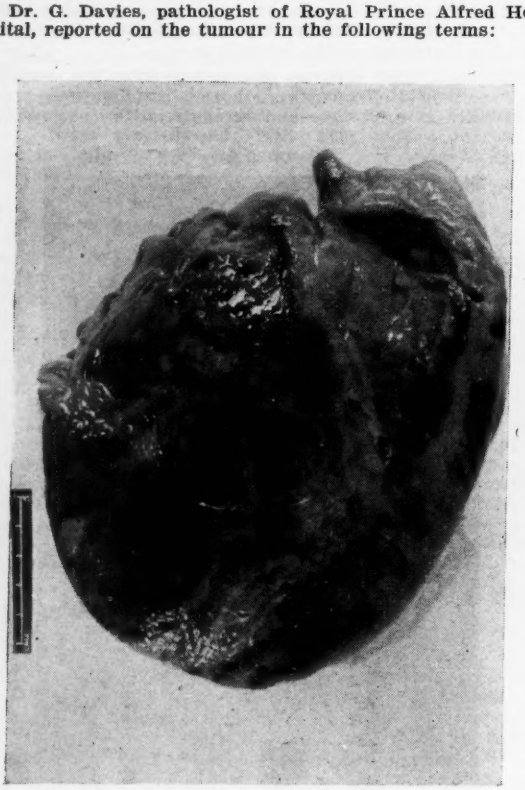

FIGURE III.

Macroscopic: The specimen consists of a very large tumour from the region of the suprarenal gland. It has been bisected in the theatre and the two portions, together with some smaller fragments of similar tissue, weighed 1070 grams. The largest piece of tissue measures about 19 × 14 × 4·5 cms. and the smaller 16 × 13 × 5 cms. The cut surface of the tumour has a somewhat variegated appearance. [Figure III.] The best preserved portions have a pale-reddish-brown colour, but on this there are very numerous areas of hæmorrhage from pin-point size up to over 1 cm. in diameter and some of these larger hæmorrhages are grouped together to form large hæmorrhagic areas. On the cut surface many large thin walled but well formed blood vessels are seen. They somewhat resemble the large veins seen on the cut surface of a liver. The tumour is covered by a smooth membrane but in parts this is lumpy as though the tumour were projecting through weak points in the outer covering. The tumour appears to have been slightly lacerated during removal.

Microscopic: The tumour is a highly cellular one, the cells being large and polygonal with abundant cytoplasm. With hæmatoxylin and eosin this is mainly pale and faintly eosinophilic but there are occasional cells which are much more deeply stained by eosin. The nuclei are mainly spherical with well formed nuclear membranes, finely stippled chromatin and frequently, large nucleoli. A few pyknotic nuclei are present and very large and hyperchromatic nuclei are fairly numerous. Those cells which stain most deeply by eosin have nuclei which are usually somewhat pyknotic. The cells occur in bread continuous sheets. In many large areas blood vessels are inconspicuous but in other parts they are numerous, large and thin walled and resemble sinusoids. They are supported by a minimum of connective tissue.

In sections stained by Vines' method most of the cells are stained a slaty-violet colour but a fairly large number have bright cherry-red cytoplasm. These cells are not granular but even under the oil immersion lens they appear to have homogeneous cytoplasm. Nuclei in these bright-red cells are mainly somewhat smaller and more pyknotic than the slaty-violet cells. It is difficult to assess the degree of malignancy this tumour may have. It has grown to a large size without destroying a thin covering membrane and the very thin walled veins seen macroscopically are not obviously invaded by tumour cells. There is a complete absence of lymphocytic or fibrous tissue reactions. All these features suggest that the tumour is not malignant. Nevertheless the nuclear hyperchromatism and irregularity suggest the possibility that some degree of malignancy may be present.

When written to later, the patient replied as follows:

> Dear Doctor, you ask was the operation worth while. My one and only regret is that it did not take place years ago. The change has been truly wonderful. I now weigh twelve stone, rolls of fat everywhere; the chest, abdomen and axillas are very clear and elsewhere a great improvement. My flesh is snowy white. Great expression has come back into my face, which is round and fat. People say I look years younger and something superhuman must have happened in Sydney. When I look back on my unhappy life of suffering, what a difference it now makes. The hair of the head is now growing and on the top shows signs of life. I have a great appetite, in fact I eat too much. As for the neuritis which troubles me that is very painful at night, but my sisters also suffer that way.

She was next examined in June, 1950, and looked well. She had maintained her increased weight. An X-ray examination of the chest revealed no evidence of metastases, and an excretion pyelogram was reported on as follows:

> On the right side the density of the kidney has increased considerably and some calyceal hydronephrosis can be seen.

She wrote subsequently, in February, 1951, in much the same terms as those of the letter quoted above.

Comment.

The case is of interest from the length of history, the size of the tumour, the high urinary excretion of 17-ketosteroids, and the apparently fortuitous association of a carcinoma of the breast, following the removal of which she has survived for five years.

SOLITARY ECTOPIC PELVIC KIDNEY.

By IAN F. POTTS, M.S., F.R.C.S.,
Sydney.

GENITO-URINARY abnormalities are the commonest of all congenital malformations. Some are relatively common in a large urological department and excite no special interest —for example, double ureters, bifid pelvis, polycystic kidneys *et cetera*. Some other genito-urinary abnormalities are very uncommon. Stevens (1937) estimated the frequency of true ectopic pelvic kidneys to be one in 2150 to 3000 autopsies, and that of a single ectopic kidney to be one in 700 to 1600 autopsies. This gives a frequency of single ectopic pelvic kidney of one in 22,000. Longo and Thompson (1952) state that unilateral renal agenesia is found in about one in 1000 autopsies, and in about one in 1500 clinical cases. This agrees fairly well with the above-mentioned frequency given by Stevens, and is much greater than is usually thought. Longo and Thompson emphasize the common association also of abnormalities in the genital tract.

The literature on solitary pelvic ectopic kidney has been adequately reviewed recently. Stevens (1937) recorded two cases and reviewed 25 previous publications, from the first description by Hanot in 1830. McCrae (1942) and Hanley and Steel (1947) brought the series to 44. Golden *et alii* (1951) brought the series up to date, but their case was not the fifty-ninth, as those of Goodhope and of Goldberg and Fowler had been previously listed. Cases since then have been described by Butler (1951), who also quotes the case of Hanbury (1949), the case of Anderson and Robertson (1952), and a case report in *The New England Journal of Medicine* in 1952.

I hesitate to label the present case as the sixty-third case, as most previous reviewers have missed contemporary cases, and I have most probably also done so.

The condition is thus of sufficient rarity to warrant the report of a further case.

Clinical Record.

The patient, C.H., a male, aged forty-five years, a refrigerator mechanic, was referred to the surgical outpatient department at Sydney Hospital in August, 1952, where he was examined by Dr. P. W. H. Grieve. He complained of acute severe left-sided backache present since June, 1952, which radiated down the back of the left leg to the ankle. During the course of his investigation a rectal examination was performed and the following comment was made: "A firm, solid mass, 6″ × 3″, of lenticular form, not tender, apparently fixed to left side of superior aspect of sacrum, ? horseshoe (pelvic kidney)."

He was referred for excretion pyelography and X-ray examination with a barium enema. The report on the latter was normal. The excretion pyelogram was reported on as follows: "The patient is ill-prepared and the urogram useless. The dye has reached the bladder and this organ appears displaced slightly to the left by a soft tissue mass in the right side of the pelvis. Gross spondylitis osteoarthritica in the lumbar spine." This examination was repeated and the following report made: "One kidney lies within the pelvis in the mid-line; it has a short ureter which joins the bladder on the right side. The left kidney appears to be normally placed, but its outline and the structure of its collecting system is again hidden by bowel content. A retrograde examination appears to be necessary."

At this juncture he was referred to the urological outpatient department. He denied any urinary symptoms. General examination revealed no abnormality. His blood pressure was normal. Neither kidney could be palpated in the loin. The external genitalia were examined carefully and were normal. Microscopic examination of a centrifugalized specimen of urine was normal and attempted culture was unsuccessful.

A cystoscopic examination was performed on November 25, 1952, under local anæsthesia. Mild trabeculation was present in the bladder. The right half of the trigone and ureteric orifice was normal. The left side of the trigone and ureteric orifice was absent. The posterior part of the urethra was normal, and no ectopic ureteric orifice was seen. A bulb catheter was introduced into the lower right ureter and a retrograde pyelogram was taken. The retrograde pyelogram confirmed the presence of a right pelvic kidney, lying over the mid-sacrum. The pelvis was directed medially, the collecting system appeared normal, and the ureter was very short. In the pyelogram the last-mentioned was slightly dilated owing to overfilling (Figure I).

When the excretion and retrograde pyelograms were reviewed, it was thought that the left kidney was absent.

A translumbar aortogram with the patient under "Pentothal" anæsthesia was taken on December 10, to substantiate this belief. The first and second pictures taken during this investigation—the "aortogram" and "nephrogram" respectively—are reproduced (Figures II and III). The aortogram is taken during the injection of the last three millilitres of 20 millilitres of 70% diodone solution, and the nephrogram as soon as possible afterwards. This is approximately a four-second interval. In the aortogram diodone has already diffused into the capillaries and tubules, and the kidney is shown as a uniform opaque mass. The aorta and its divisions are well seen. The aberrant blood supply to the kidney cannot be identified (Figure II).

In the nephrogram all the dye has passed out of the arterial tree, but sufficient remains within the kidney substance to outline the organ clearly. The hilum is well seen on the inferior border in the middle portion. Neither a kidney shadow nor a renal artery can be seen in either loin in either film (Figure III).

Thus the aortogram substantiated the diagnosis of a right pelvic ectopic kidney and the absence of a kidney on the left side.

A rectal examination was performed whilst the patient was under the anæsthetic. The kidney was easily felt, the lower pole being most accessible. A finger could be introduced behind this and the posterior aspect felt over about half its extent. The contour, dimensions and consistency felt normal. It possessed a limited mobility. At a later date, when the patient was conscious, movement of or pressure upon the kidney with a finger inserted in the rectum had no effect on his sciatica.

This backache was considered to be due to a prolapsed intervertebral disk, and not related to pressure on his sacral nerves by the pelvic kidney. He was referred to the orthopædic department, where he was treated as suffering from a ruptured intervertebral disk.

Thus his renal anomaly was discovered in the course of investigation of an unrelated disability, by a first-class piece of clinical diagnosis.

Discussion.

The solitary pelvic kidney is compatible with good health, but is more prone to stasis, infection, stone and (some maintain) tuberculosis. Owing to its failure to ascend, it is always malrotated, usually with the pelvis lying anteriorly, and it has an abnormal arterial supply. These arise from the lower part of the aorta and the iliac vessels, and are variable in number, origin and course. A very real danger is that it may be removed unwittingly as an extraperitoneal mass at an exploratory laparotomy, as was done by Polk in 1883 and by Strube in 1894, with fatal results. The latter case is of further interest, in so far as death was delayed for a month. In the event of pregnancy, Anderson *et alii* (1949) state that "most cases of unilateral ectopic pelvic kidneys associated with pregnancy will deliver spontaneously provided the patient has an adequate pelvic capacity".

Associated genital lesions are very common. In females absence of the vagina, uterus or Fallopian tubes is very common. In one series, in 17 of 19 cases the vagina was absent. Golden states that in 55% of cases concomitant genital abnormalities are present, usually on the same side.

Males may show absence of or imperfect descent of testes, or absence of the epididymis.

The embryology of the condition has been adequately discussed previously (Fortune, 1928; Butler, 1951). The agenesis seems most probably due, as suggested by Fortune, to (i) absence of metanephrogenic bud, or (ii) early degeneration of the metanephrogenic bud, or (iii) improper development of the mesonephros.

The abnormal arterial blood supply is a result and not a cause of the ectopia. The associated genital abnormalities are readily understood because of the close interrelation of the Wolffian and Müllerian duct systems during development.

The trigone arises from the distal end of the mesonephric or metanephric duct, and absence of the ureter causes failure of development of the ipsilateral half of the trigone.

The condition is equally common in both sexes, and occurs with equal frequency on either side.

Summary.

1. A further case of a solitary pelvic ectopic kidney is reported. It is believed that this is approximately the sixty-third recorded case.

2. The condition was discovered, *en passant*, during the investigation of an unrelated disorder, and itself was asymptomatic.

3. Apart from the usual urological investigations, a translumbar aortogram was taken, and it is believed that this is the first occasion on which this has been done in this anomaly.

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Legends to Illustrations.

FIGURE I.—Right retrograde pyelogram. Cystoscope *in situ*. Clearer definition of the excretion pyelogram appearance. Ureter dilated owing to overfilling.

FIGURE II.—Aortogram. Needle in abdominal aorta. Outline of aorta and main divisions retouched. Outline of kidney visible owing to its complement of dye. Aberrant blood supply cannot be defined.

FIGURE IV.—Nephrogram. No dye remains in arterial tree. Kidney of increased density. Hilum present on inferior border in its middle portion.

Reviews.

Textbook of Medical Treatment. By various authors, edited by D. M. Dunlop, B.A. (Oxon.), M.D., F.R.C.P. (Ed.), F.R.C.P. (London), L. S. P. Davidson, B.A. (Camb.), M.D., F.R.C.P. (Ed.), F.R.C.P. (London), M.D. (Oslo), and John McNee, D.S.O., M.D., D.Sc. (Glas.), F.R.C.P. (Ed.), F.R.C.P.S.; Sixth Edition; 1953. Edinburgh and London: E. and S. Livingstone, Limited. 9½" × 7", pp. 1040, with 44 illustrations. Price: 50s.

THE sixth edition of this well-known text-book maintains the excellent standard set in the previous editions. This book has certainly established itself as an authoritative and progressive publication; each edition bears the stamp of thorough revision. For example, to meet current inquiry, new chapters have been added on several subjects, including the chemotherapy of malignant disease. The same broad view but critical approach is seen here as was commented on by us in the review of the fifth edition, when the then comparatively new antihistamine drugs were dealt with.

The chapter on tuberculosis is excellent. It is well arranged and well illustrated and maintains a nice balance between the conservative and obviously minimal treatment required for some cases and the more complicated procedures such as thoracoplasty and lung resection. The drug treatment of the disease is well described, though the iso-nicotinic acid derivatives are not given a great deal of attention. However, the statement that "present evidence does not support earlier claims but the drugs [that is, iso-nicotinic derivatives] may be of value in association with those other drugs whose worth has been proved" is an example of the general critical and cautious approach, and we agree that nothing more need be said or written. Despite the great detail necessary in a book of this nature, the authors do not hesitate to give advice crisply and to the point. For example, in the section on the general treatment of pulmonary tuberculosis, after naturally stressing the fundamental requirements of rest and proper feeding, they state "one need not be fussy about diet". How necessary this salutary point of view is in some quarters!

The whole book is, as was the aim expressed in the first edition, explicit, and every endeavour is made to give, where possible, unequivocal advice to any practitioner seeking guidance in treatment. For example, in the section on hyperthyroidism the (in some quarters) vexed question of thiouracil treatment *versus* operation is admirably summarized, and he would be indeed a difficult doctor to please who could not decide into which category his patient fitted.

We repeat one criticism of the last edition: if the authors (we think rightly) prefer the metric system in dosage, why do they still refer to "hæmoglobin percentages" instead of the almost universally used figures expressed in grammes?

Notes on Books, Current Journals and New Appliances.

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A SPECIAL FEATURE of the September issue of *Family Doctor* is a School Section containing readable articles on various aspects of the mental and physical welfare of the school child. Some details in these articles apply to the English scene, but the intelligent parent of any country will find much useful information and food for thought in the section. In the section for family reading is a practical article on how to keep warm and two well-established features, "Close-up of Science" and "How Your Life Began". Medical articles deal with swollen ankles, poliomyelitis, perfectionists, preparation for breast-feeding and a sensible article written in answer to a reader's question about a naughty child. Briefer answers to questions under the heading "Expert Advice" are becoming an important feature. Other interesting subjects dealt with are summer diet and fruit-bottling. The high standard of illustration and general presentation is well maintained.

Any reader who would like to receive a specimen copy of *Family Doctor* is invited to send a request to the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, who will send on his request to England.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Pocket Obstetrics", by Arthur C. H. Bell, M.B., B.S., F.R.C.S., F.R.C.O.G., Hon. M.M.S.A.; Third Edition; 1953. London: J. and A. Churchill, Limited. 7½" × 5", pp 164, with 14 illustrations. Price: 8s. 6d.

The first edition was published in 1946.

"The 1952 Year Book of Pathology and Clinical Pathology (January-December, 1952)"; Pathology, edited by Howard T. Karsner, M.D., LL.D.; Clinical Pathology, edited by Arthur Hawley Sanford, M.A., M.D.; 1953. Chicago: The Year Book Publishers. 8" × 5¼", pp. 400, with 164 illustrations. Price: $5.50.

One of the Practical Medicine Series of Year Books.

"A Ciba Foundation Symposium: The Spinal Cord"; consulting editors, J. L. Malcolm, M.B., Ch.B., B.Med.Sc., J. A. B. Gray, M.A., M.B., B.Chir., G. E. W. Wolstenholme, O.B.E., M.A., B.M., B.Ch., assisted by Jessie S. Freeman, M.B., B.S., D.P.H.; 1953. London: J. and A. Churchill, Limited. 8" × 5¼", pp. 312, with 112 illustrations. Price: 30s.

Presents the latest views on the anatomy, physiology and pharmacology of the spinal cord.

"A Manual for Psychiatric Case Study", by Karl A. Menninger, M.D.; 1952. New York: Grune and Stratton. London: George Allen and Unwin, Limited. 9" × 6", pp. 368. Price: 50s.

Intended specially to meet the needs of doctors entering the field of psychiatry.

"Annual Review of Medicine", edited by Windsor C. Cutting and Henry W. Newman; 1953; Volume IV. Stanford, California: Annual Reviews, Incorporated. 9" × 6¼", pp. 462. Price: $6.00.

Contains eighteen carefully documented articles and also an "annotated list of reviews in medicine".

The Medical Journal of Australia

SATURDAY, SEPTEMBER 26, 1953.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

IMMUNIZATION AGAINST DIPHTHERIA, WHOOPING-COUGH AND TETANUS.

THAT it is possible to protect persons against disease by either active or passive immunization has been known for many years. Active immunization may be carried out by the inoculation of attenuated living organisms or viruses or by inoculation with vaccines of killed organisms prepared from cultures of the appropriate organism in the appropriate phase. An example of the use of attenuated living organisms is the use of calf lymph in protection against smallpox and more recently the use of BCG vaccine for protection against tuberculosis. Probably the best known vaccine of killed organisms is TAB vaccine prepared from cultures of *Salmonella typhi*, *Salmonella paratyphi* A and *Salmonella paratyphi* B. In recent years the use of toxoids has been added to the measures which may be used to protect against disease. Bacterial toxins may be altered by treatment with formalin so that they are no longer toxic but are still antigenic. Such altered toxins are called toxoids. In most countries today the use of one or other of the many forms of diphtheria toxoid is advocated on a national basis by the appropriate public health authority, and the reduction in the incidence of diphtheria has been remarkable. The use of *Hæmophilus pertussis* vaccine to give protection against whooping-cough is now also strongly recommended by many health authorities, although the protection afforded by the administration of such a vaccine is perhaps not as high as that obtained with toxoid against diphtheria.

Investigations to test the value of pertussis vaccines have been carried out under the auspices of the Medical Research Council of Great Britain since 1942 and are still in progress. Field trials have proved the efficacy of these vaccines. Field trials are cumbersome and time-consuming and investigations are at present being carried out in Great Britain to devise a means of assaying the potency of a vaccine by laboratory tests. In these tests mice are immunized, and the level of protection acquired is tested by an intracerebral challenge injection of virulent *Hæmophilus pertussis*. It has not yet been determined whether such a procedure will afford an accurate means of determining the ability of a vaccine to confer protection on children. During World War II immunization against tetanus was carried out in all services by the inoculation of all personnel with tetanus toxoid in three injections—the two first injections at a six-week interval and the third injection one year after the second. That such a procedure was effective was shown by the extremely low incidence of tetanus in Allied troops compared with that in the enemy troops who were not so immunized. The use of tetanus toxoid on a national basis has not yet been advocated by health authorities, chiefly because the incidence of tetanus is in no way comparable with the incidence of diphtheria and whooping-cough. There is no doubt that persons at risk should be immunized against tetanus, and it might well be that the time is now at hand to institute an immunization campaign against tetanus on the same basis as that advocated for diphtheria and whooping-cough.

Immunization against diphtheria, whooping-cough and tetanus may be carried out by injecting the antigens either singly or in the combined form. There are still two schools of thought whether single antigens or combined antigens should be used. Combined antigens are used widely in the United States of America and to some extent in Great Britain. In a recent issue of the Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service directed by the Medical Research Council of Great Britain it is stated that a combined diphtheria-pertussis vaccine which ensures a high degree of immunity against both infections is much to be desired since the number of injections would be reduced. Recent immunological research on animals by Barr and Llewellyn-Jones shows, however, that the devising of such a substance may be a much more complicated matter than hitherto believed. Their preliminary report suggests that the effect of immunization with combined antigens may depend on the relative proportions of the components, the dosage administered, and the immunity state of the population injected. This is clearly a matter which requires further elucidation before combined immunization can be recommended without reservation.

There has been a change of mind about the age at which immunization should be carried out. There is increasing evidence that the very young infant is capable of responding to antigens with the production of antibodies when passive immunity does not exist. There have been favourable reports when antigens have been administered as early as the third month. The Committee on Immunization and Therapeutic Procedures for Acute Infectious Diseases of the American Academy of Pediatrics has published its recommendations for immunization with injections of combined antigens at as early an age as three months.[1] It is well recognized that "booster" doses of the toxoids are necessary, but the time interval of such

[1] *New England J. Med.*, December 11, 1952, page 941.

"booster" doses is still a matter of opinion. It is generally conceded, however, that it is advisable to give at least two "booster" doses of diphtheria toxoid at approximately three and six years after the primary injection. It is also thought that it is probably necessary to give "booster" doses of tetanus toxoid at five-year intervals after the completion of the first course.

McCloskey has shown that in patients who receive an injection of antigen during the month prior to onset of poliomyelitis there may be a close relationship between the site of paralysis and the site of injection. These reports have been confirmed in England and America. Such a relationship is not apparent in persons who receive the injection of antigen more than a month prior to the onset of the disease. The question is still under investigation, but in view of these reports it might be advisable to suspend injections of antigens during a poliomyelitis epidemic. It is a matter for grave thought whether such a suspension should apply to diphtheria toxoid. A widespread epidemic of diphtheria would have much greater consequences than scattered cases of poliomyelitis in which paralysis occurred in an injected limb.

Current Comment.

ANTICOAGULANTS IN MYOCARDIAL INFARCTION.

IT is interesting to look back over the history of anticoagulant drugs in the treatment of myocardial infarction and to observe the pendulum movement of opinion from scepticism to wild enthusiasm and now back to modified acceptance. The medical world is used to watching such oscillations, and it is always fascinating to see where the swing will stop. It is salutary, too, to realize that anticoagulant drugs have risen high on the list of drugs causing deaths, as N. Flaxman has shown,[1] and yet we are still quite uncertain of their true value in coronary heart disease, eight years after I. S. Wright first reported using them.[2] This blot on the history of medical science stands in striking contrast to the speed and precision with which the Medical Research Council in Great Britain has assessed the value of streptomycin, PAS and isoniazid in tuberculosis. The comparison pays great tribute to the principle of controlled therapeutic trials, and proper trials are sorely needed for the anticoagulant drugs.

If some new treatment revolutionized the outlook for patients suffering from myocardial infarction, then its value would be obvious to all and strictly controlled trials would not be needed. If, on the other hand, we were dealing with a disease which was uniformly fatal or which ran some other easily predictable course, then it would be relatively easy to assess the influence of a new form of treatment. But anticoagulants are not obvious "winners", and myocardial infarction is a disease which defies prediction. D. A. Rytand,[3] who has written one of the most critical of the recent reports expressing dissatisfaction with the position of anticoagulants in treatment, points out that the mortality reported for different series of cases even without any special treatment varies from 8% to 78% and the incidence of thrombo-embolism from 11·5% to 42%. A low mortality attributed to anticoagulants by some observers would be regarded as an unduly high mortality for a series of untreated patients by other observers. It is quite clearly fallacious to compare the results in one group of patients treated with anticoagulants with the results in any other group of untreated patients reported from elsewhere or obtained in previous years. This is what has been done in the majority of instances. The groups must be strictly comparable in respect of severity, age, sex, other forms of treatment and anything else which might influence mortality. A strictly controlled trial is the best way to achieve this, and only one such trial has been reported. In this trial L. Feldman and his colleagues[4] treated alternate patients with anticoagulants, demonstrated that an adequate level of anticoagulation was achieved and showed that the two groups were similar in age, sex and so on. They found no differences either in mortality or in thrombo-embolic complications: in each group of 76 patients, 23 died. Most of the other so-called trials which have been reported are open to criticism. For example, I. S. Wright *et alii*[5] state that the American Heart Association, which reported a mortality of 16% as opposed to 23% in favour of anticoagulants, collected the results from a number of hospitals in which patients were given anticoagulants or not according to whether they entered hospital on odd or even days. But Ryland points out that the difference in numbers of patients in the two groups is greater than would be expected from a strict adherence to this method of allocation and gives other evidence to show that treatment was sometimes given out of turn. A flaw like this destroys the whole basis of clinical trials, makes statistical analysis invalid and reduces the results to the category of "suggestive but not proven". Another example of a trial critcized by Ryland is that of F. J. Schilling,[3] who reported case fatality rates of 16·7% and 40% in groups treated with and without anticoagulants respectively. The mortality of the combined groups was 28%, while for similar patients without treatment at the same hospital in previous years it was 26%. Ryland suggests that a legitimate conclusion would be "that the prognosis for a patient with myocardial infarction is worse when anticoagulants are given to someone else".

The history of this subject is strewn with many such reports which are suggestive but inconclusive of benefit being derived from anticoagulants. One of the more recent and better articles in support of this form of treatment comes from Oxford. I. S. L. Loudon, J. C. Pease and A. M. Cook[4] have not conducted a trial, but have reviewed their records in better style than many authors. They have shown that their treated and untreated patients agree reasonably well in respect of age, sex, severity of shock and cardiac function, and they conclude that the difference in mortality between the two groups (25% as opposed to 40%) favours the use of anticoagulants. They suggest that treatment is of most benefit to patients over the age of sixty years, moderately or severely shocked, and with little or no previous impairment of cardiac function. There is a good deal of support for restricting the use of these drugs to the most severely ill patients, with the emphasis on severity rather than on age, as H. I. Russek and B. I. Zohman[5] have shown.

The picture becomes even more puzzling if we turn to consider thrombo-embolism. Here, where we would expect the effect of anticoagulants to be greatest, results are less impressive than the reported reductions in mortality. Various explanations are put forward to account for this—that anticoagulants counteract shock (A. R. Gilchrist[6]), that they increase coronary blood flow (N. C. Gilbert and L. A. Nalefski[7]), and that the benefit derived from heparin is due in part to its effect on lipoprotein metabolism (D. M. Graham *et alii*[8]). There is at least one very clear and significant piece of work which has been reported recently. H. P. Wright, M. M. Kubik and M. Hayden[9] produced thrombosis of the femoral artery in rabbits and followed the course of recanalization by arteriographic studies and

[1] *J.A.M.A.*, September 29, 1951.
[2] *Proc. Am. Fed. Clin. Res.* (1945), 2:101.
[3] *Arch. Int. Med.*, August, 1951.
[4] *Am. Heart J.*, July, 1952.
[5] *J.A.M.A.*, December 11, 1948.
[6] *J.A.M.A.*, July 1, 1950.
[7] *Brit. M. J.*, April 25, 1953.
[8] *Am. J. Med.*, June, 1953.
[9] *Brit. M. J.*, August 16, 1952.
[10] *J. Lab. & Clin. Med.*, June, 1949.
[11] *Circulation*, November, 1951.
[12] *Brit. M. J.*, May 9, 1953.

final histological examination. When rabbits were treated with "Tromexan" the arteries were recanalized in about three or four weeks, whereas in the controls the arteries were still occluded after several months. It would seem that the clinicians have had their fling, and we are back to the animals with very pretty proof of the value of anticoagulants in arterial thrombosis.

A study of the literature relating to the use of anticoagulant drugs in myocardial infarction highlights three facts: (i) myocardial infarction is a dangerous disease, (ii) anticoagulants are dangerous drugs, and (iii) how far the dangers of the disease are reduced by using these drugs is not known with certainty. We are not acting unwisely if we restrict their use to severely ill patients and control the level of anticoagulation rigorously.

EMPLOYMENT OF DISABLED YOUTHS.

THE year 1944 saw the passage in Great Britain of the *Disabled Persons (Employment) Act*. This Act provided that the Ministry of Labour should establish and maintain a voluntary register of disabled people, and that the larger firms should be obliged to employ a quota of workers who were registered in this way. The Act also gave the Ministry power to set up units to train or rehabilitate the disabled. At the request of the Ministry of Labour the Medical Research Council set up an Occupational Medicine Committee in order that information might be obtained about the medical problems of placing disabled people in employment. This committee has now issued a memorandum which is published by the Medical Research Council. The memorandum is written by T. Ferguson, A. N. Macphail and Margaret I. McVean.[1]

In the preface, the report is summed up by the statement that the committee has explored the employment experience of two groups of young people in Glasgow. One was a group of registered disabled and the other was a group of children who had been to special schools for the physically handicapped and who, for various reasons, had a registration rate of only 30%. The investigation, we are told, illustrates not only the vicious circle of illness—loss of schooling followed by unskilled, often heavy work and then further illness—from which the disabled find it so hard to escape, but also the difficulty of ensuring that registration helps those who most need help. In the investigation of the first group some 579 disabled young people, who had been registered (the total number was 683, but 65 could not be traced), were sent a preliminary letter and were interviewed in their homes; three asked to be seen elsewhere as they were "ashamed of their home conditions". In general, the cooperation secured was excellent, particularly from parents, who were anxious to receive advice and information. The disabilities, which are set out in a lengthy table, included the infective and parasitic diseases, neoplasms, general diseases, such as asthma and diabetes, diseases of the blood, mental and psychoneurotic conditions (chiefly mental retardation), diseases of the nervous system (congenital paralysis and epilepsy, deaf-mutism and defective vision providing the greater number), diseases of the circulatory system (chiefly rheumatic carditis), diseases of the respiratory system, diseases of the digestive system, diseases of the skin and cellular tissues, congenital malformations, diseases of bone and organs of movement, diseases of early infancy, ill-defined conditions and accidents through poisoning and violence. The second group included 408 young people who left schools for the physically handicapped during the period 1945 to 1948. The outstanding difference between the groups was that whereas only some 30% of those who attended special schools for the physically handicapped were registered as disabled, all members of the other group were so registered. The basis of admission to a special school is one of educational need, whereas the basis of admission to the register of disabled persons is a "substantial handicap" in obtaining and holding suitable work. It should perhaps be pointed out that the disabilities in the group of 408 children were much the same as those of the other series, except that mental and psychological conditions were excluded. It is stated that the nature and severity of the disability inevitably exerted a great influence on the employment pattern. Each disabled lad presented his own individual problem. This was often heavily coloured by social background. In each case careful individual consideration was needed. The difficulties experienced by some of the young people ranged from those of the cardiac cripple to the classical miseries of epilepsy. It is also pointed out that it may be very difficult for severely disabled young people such as those discussed to translate the prescription of light work from the sphere of what is ideal to the sphere of what is practicable. The same kind of dilemma perplexes the tuberculous youth who has to avoid heavy work and needs plenty of fresh air, but who discovers that fresh air is accompanied by heavy work outdoors, and light work by a stuffy atmosphere indoors. When a child first leaves school, his attitude to employment is usually positive and full of enthusiasm. No better time could be chosen for his taking up suitable work. It is stated that too often constructive action is delayed, and that as months pass the youth drifts into unskilled manual labour. At first, this is comparatively well paid and the youth resents advice which urges him to take a job which may be skilled but which brings him in for the time being a smaller wage. This, of course, is a feature of present-day life in most countries; it certainly is in Australia.

In the conclusions of the study it is stated that apart from the overriding need to improve basic living conditions, particularly housing, and to harness all the resources of medicine for the prevention of disease, the findings of the study suggest four lines of action which seem to hold promise of substantial improvement. The first is mitigation of the gross educational lack that adds to the difficulties of many of these young disabled people. This is obvious, and all will agree that the educational training of a child with a residual disability (as, for example, a disability following poliomyelitis) should be decided upon after the most careful consideration of individual needs. In Australia, the educational departments of the several States are alive to this problem, and a great deal is done to provide education for the disabled. The second line of action is the adoption of energetic steps to secure satisfactory settlement before the young person begins to drift. This has yielded rich dividends in Glasgow in regard to deaf children. The ideal time for successful settlement is when the child leaves school. If the child can be given sufficient insight into his condition and be made to realize that there is a purpose in life, he will regard his disability as a challenge and may in the long run do better than other children who have no disability but have acquired no insight. The third line of action mentioned is the provision of trainee centres for disabled young people, and the fourth is the provision of intensive medical and surgical treatment when necessary, together with the provision of any necessary medical aids or surgical appliances. These two aims require no further comment.

Short reference may be made in conclusion to work which is being carried out in Denmark. During the years 1938 to 1944, some 10,000 notifications of "invalidity" were received by the Danish Invalidity Court, and during the same years, 3167 disabled Danes received vocational measures under the auspices of the Court. Of this total, 1296 had started training before the age of eighteen years. Though precise information about the age distribution of notified persons is not available, virtually all were under the age of thirty years. We read that it seems reasonable to conclude from available data that the proportion of disabled young people in Denmark who receive vocational training is considerably higher than in Great Britain. Fortunately, in Australia a good deal is done for the rehabilitation of the disabled. Readers may remember a paper by Dr. Douglas Galbraith, which was presented at

[1] "Employment Problems of Disabled Youth in Glasgow", by T. Ferguson, A. N. Macphail and Margaret I. McVean; Medical Research Council Memorandum No. 28; 1952. London: Her Majesty's Stationery Office. 9¼" x 6", pp. 72. Price: 3s. net.

the Sixth Session of the Australasian Medical Congress (British Medical Association), held at Perth in 1948. Galbraith described what is being done. He concluded his survey with a statement that if a wider content of medicine was put into practice in Australia, we might reasonably expect diminution in the number of unemployed disabled people. "We could also anticipate a great decrease in the ranks of those peripatetic—and often pathetic—chronic inhabitants of the out-patient departments of our public hospitals."

PEMPHIGUS.

ALTHOUGH pemphigus means simply a condition characterized by the appearance of bullæ on the skin, it is not accepted as including all bullous conditions. It has been used to describe many conditions of the skin from the time of the Hippocratic writings onwards, but its present-day usage seems to date from the latter half of the eighteenth century. Walter F. Lever[1] cites Wickmann, in 1791, as giving the word its present meaning, that of a chronic bullous disease. Many subsequent departures were made from this basic concept, but most authorities on dermatology now confine the term to a fairly well-defined small group of conditions. The four listed as true pemphigus by Lever are *pemphigus vulgaris malignus*, *pemphigus vegetans*, *pemphigus foliaceus* and *pemphigus erythematosus* (Senear-Usher); a second group, classed as "pemphigoid", includes bullous pemphigoid (? bullous *dermatitis herpetiformis*) and benign mucous membrane pemphigoid (*pemphigus conjunctivæ*). Opinions vary on just how far the conditions listed by Lever as true pemphigus are distinct, but his list corresponds in many respects to that of other writers, with the notable exception that he separates *pemphigus vulgaris* into two types: *pemphigus vulgaris malignus* and bullous pemphigoid, a benign form which he then rejects from the true pemphigus group. His view of acute malignant pemphigus (the so-called butcher's pemphigus), which, despite its name, is not accepted by most authorities as true pemphigus, is that it is either *pemphigus vulgaris malignus* running an unusually rapid course or severe *erythema multiforme*. This is all intended to help towards a more accurate classification, but it must be felt by many that the position is still somewhat confused. It is interesting to note that Sequeira, Ingram and Brain, in the fifth edition of their well-known text-book published in 1947, describe together in one chapter "a group of diseases with fairly well-defined characters in which the formation of blisters is the predominant feature"; they include acute malignant pemphigus, *dermatitis herpetiformis*, *hydroa gravidarum* and the various forms of "true pemphigus" already listed, and they make the following illuminating comment: "It is not possible to classify them satisfactorily in the present state of our knowledge, and in a number of cases the relationships are so indefinite that many careful observers prefer to apply the name 'bullous eruption', which has the advantage of being non-committal."

Lever makes his classification firmly, selecting his four types of true pemphigus because they have the histological picture of pemphigus described as characteristic by Civatte in 1943; its features are acantholysis and intraepidermal bulla formation. In the two types of pemphigoid described by Lever no acantholysis occurs and the bullæ form subepidermally. Thus he regards histological examination as very important in differential diagnosis. So far as the result of the pathological picture is concerned, there seem to have been no constant changes found *post mortem* in the internal organs of subjects who have died of pemphigus. Effects observed during life include severe anæmia and hypoalbuminæmia in patients with *pemphigus vulgaris malignus*. The concentration of albumin in the blood serum, determined by electrophoresis, may decrease to one-fourth of the normal value. The bullous fluid contains considerable amounts of protein averaging 70% of the concentration present in the corresponding blood serum. On electrophoretic analysis, the relative amounts of the various proteins are very similar in the bullous fluid and in the corresponding blood serum.

Many suggestions have been offered on the ætiology of pemphigus. Lever has patiently reviewed them, but the position is summed up in his terse statement: "The cause of pemphigus is unknown and not even a convincing theory as to its cause exists." With a little qualification a similar statement might have been made about treatment until recently. Many forms of therapy have been favoured, but the results have been in general disappointing. The mortality rate was disturbingly high. With the advent of ACTH and cortisone this has been altered. The effect of these drugs is suppressive, as it is with most of their uses; but, according to Lever, all types of pemphigus and pemphigoid respond well, provided adequate doses are administered. Although the improvement is temporary, judicious administration of these drugs either in intermittent courses or in continuous doses has been found to keep the disease in remission and so to make possible the saving of the lives of patients with the malignant form of the disease. The essential would appear to be adequate dosage. Lever discusses details of therapy in the light of experience and presents an impressive account of results obtained. In a series of 23 cases, every patient responded to treatment with ACTH and cortisone. Lever points out that although treatment with these drugs entails some risk, for patients with a life-threatening disease this risk is worth taking; and, in fact, no fatality due to the disease or to its treatment has occurred in the series. The question of cure in any strict sense is not yet decided. It is not yet known if *pemphigus vulgaris malignus* is a self-limited disease. That it may be self-limited is suggested by the fact that one of the four patients with *pemphigus vulgaris malignus* in the series has been free from lesions for over one year and another for four months. If this hitherto often mortal disease should prove to be self-limited in most patients, then a notable therapeutic victory would have been won; in other words, treatment with ACTH or cortisone, while not curative in an absolute sense, would represent (in Lever's words) not merely a palliative but actually a life-saving treatment for pemphigus.

[1] *Medicine*, February, 1953.

FIDGETS IN THE LEGS.

ANYONE who has suffered from fidgets in the legs will agree that the condition is most irritating and difficult to treat. A person may be perfectly normal to all appearances when he is going about his daily duties, but as soon as he goes to bed and becomes warm his legs refuse to keep still. Try as he will, he cannot control them, and in sheer desperation he is forced to get out of bed and walk about. Mental irritation probably adds to the trouble. The condition has been described as "jittery" legs, but the word "fidgets" seems the more appropriate. Years ago, Robert Scot Skirving contributed an article to this journal: "On Herpes, Cramps, Fidgets and Other Disordered Sensations."[1] One paragraph from his paper should be quoted:

> Fidgets, out of bed, are dependent on psychical conditions as well as physical. Sitting on an uncomfortable chair in a cold and stuffy room and bored stiff with your company, you are just the *milieu* to find yourself with them. I have even known them to occur in a church. In bed matters are still worse. They banish sleep and I do not doubt that they must sometimes be an active source of connubial unrest. I dare say the same causes which produce cramps in one person may lead to fidgets in another; but some fidgets are certainly related to constitutional states and food, for I have seen gouty subjects who had sleep-killing fidgets relieved by colchicum and lithia.

The subject is brought to mind by the publication of an article entitled "Therapy in Restless Legs" by Nils

[1] M. J. AUSTRALIA, May 9, 1936.

Brage Nordlander.[1] He states that the condition exists in a more or less severe form in about 5% of a normal population and adds that it is seldom dealt with in medical literature. The reason for this, he thinks, is that the disease is not a deadly one; most patients never visit a doctor for it, and if they visit a doctor for another reason they do not mention their restless legs until directly asked about them. Nordlander describes the syndrome as a sort of paræsthesia, a crawling sensation felt deeply in the legs, "in the very bone marrow", sometimes amounting to real pain. He has tried various remedies and has been successful with some of them. He has used salicylates and barbiturates, and also vasodilating drugs. In 1951, he found three patients who suffered from restless legs and they all had anæmia of varying causation. The symptoms disappeared when blood transfusion was given. In cases of sideropenia in which no anæmia was present, cure has been effected by iron therapy *per os*. Since then Nordlander has tried intravenous iron therapy with "Intrafer". This is identical with "Ferrivenin" and contains a 2% colloidal solution of saccharated oxide of iron. He has also used this drug with success in patients suffering from restless legs who had no anæmia and had a normal serum iron content. Nordlander points out that it is impossible to decide the way in which treatment works as long as the pathogenesis of the condition is unknown. He thinks it possible that the fate of the colloids in the organism can throw some light upon it. He states that colloids become attached to reticulo-endothelial cells and are stored there for several weeks. If the therapeutic effect depends upon a blocking of the reticulo-endothelial system, he thinks that that could explain the long-standing improvement after one or two injections. He is undertaking investigations in order to test this possibility.

HYPERVITAMINOSIS A.

WITH the manufacture on a large scale of the vitamins, their exhibition in attractive and easy-to-take tablets and capsules and their relatively low price, together with much wrongheaded propaganda, there is a great deal of excess consumption of vitamins. The sale of vitamins in the United States of America during 1947 amounted to $188,000,000. A 300% increase in the sale of costly high potency preparations occurred in the United States between 1945 and 1947. Belief that vitamins, additional to those in the diet, improve health, increase resistance to infection and hasten convalescence from any type of illness is firmly held, not only by members of the general public, but also by many medical men, in spite of a vast amount of careful work which does not support these beliefs. In relation to only two of the vitamins is there at present definite evidence of the bad effects of overdosing; these are vitamin *A* and vitamin *D*. A review of the effects produced in man as a result of excessive intake of vitamin has been published by A. G. Knudson and P. E. Rothman.[2] It has long been known among Esquimos and Arctic travellers that the ingestion of polar-bear liver may cause severe illness. In 1942 Rodahl and Moore identified the toxic substance in the liver as vitamin *A*. Polar-bear liver may contain over 26,000 international units of vitamin *A* per gramme. Seal liver and shark liver may be equally rich. Some of the sharks in Australasian waters have livers with a very high content of vitamin *A*, up to 200,000 units per gramme, and these are used for the manufacture of vitamin *A* concentrates.

In New Zealand several cases have occurred in which severe symptoms have followed the ingestion of shark liver and also the consumption of livers of groper, kingfish and swordfish. Vitamin *A* poisoning may be chronic or acute. Chronic poisoning has been observed in a woman who took about 600,000 units of vitamin *A* per day for a period of eighteen months, supplemented by doses of 1,000,000 to 2,000,000 units on special occasions—for example, when she suspected the onset of a cold and had a sore throat. She was affected by excessive loss of hair and generalized migratory bone pains. Menstruation was abnormal, and she suffered from pruritus and dryness of the skin with some pigmentation, and from fissuring at the corners of the mouth and nasal apertures. Within ten days after vitamin therapy had been discontinued the symptoms disappeared. A number of cases have been described in children. Anorexia, pruritus and a failure to gain weight are the commonest initial symptoms. Increasing irritability, tender bone swellings, limitation of motion and resistance to handling appear. Loss and coarseness of hair, skin eruptions and desquamation, cracks at the corners of the mouth and bleeding lips are common. X-ray examination of the tender swellings reveals hyperostoses. Symptoms clear up rapidly on reduction of vitamin *A* intake. Acute hypervitaminosis *A* has occurred in a number of children from the accidental taking of large doses of vitamin *A* concentrates. In this condition vomiting and drowsiness occur, but the most pronounced feature in infants is bulging of the fontanelle.

In the adult acute poisoning occurs most frequently after the ingestion of one of the livers mentioned earlier. Headache is the predominant manifestation and is very severe, localized in the forehead and eyes. Nausea, vomiting, vertigo, sluggishness, irritability and a generalized peeling of the skin are the most commonly reported symptoms.

It has been estimated that the intake of more than 1,000,000 units of vitamin *A* is necessary to cause severe acute symptoms in adults. That milder reactions are not uncommon, particularly in infants, is very probable. Patients with unexplained bone and joint pains, headaches, pruritus, dryness of the skin and failure to gain weight and an elevated serum vitamin *A* level should be investigated for excessive vitamin *A* intake.

[1] *Acta med. scandinav.*, 1953, 145, Number 6.
[2] *Am. J. Dis. Child.*, March, 1953.

DR. GEORGE W. KOSMAK.

THE July, 1953, issue of the *American Journal of Obstetrics and Gynecology* pays a special tribute to Dr. George W. Kosmak, who has attained his eightieth birthday. Dr. Kosmak is known to obstetricians and gynæcologists the world over on account of his association with the *American Journal of Obstetrics and Gynecology*. He has been its editor for many years. He first became associated with the predecessor of this journal, *The American Journal of Obstetrics*, in 1906, and succeeded as editor upon the death of Dr. Brooks Wells in 1908. The journal had a precarious existence for a few years, but when publication of the present journal was undertaken by the C. V. Mosby Company, he became editor and acted in that capacity for more than thirty years. Obstetricians and gynæcologists all over Australia will join in congratulations to Dr. Kosmak and in best wishes.

MYASTHENIA GRAVIS AND PREGNANCY.

SOME interesting conclusions are drawn by D. Fraser and J. W. Aldren Turner[1] from a relatively large series of 14 cases of pregnancy in patients with *myasthenia gravis*. Myasthenic patients manage to go through pregnancy without especial difficulty, though increased doses of neostigmine may be needed in the first three months. Termination of pregnancy is not indicated, and normal labour can be expected. The greatest danger is relapse in the post-partum period; so labour should be conducted in hospital and the patient then stay there for three or four weeks. The child may have myasthenic symptoms, but these respond to neostigmine and usually disappear in a few weeks.

[1] *Lancet*, August 29, 1953.

Abstracts from Medical Literature.

SURGERY.

Innervation of the Bladder.

LOWRAIN E. McCREA AND DONALD L. KUMMEL (*Am. J. Surg.*, November, 1952) have demonstrated an accessory nerve supply to the bladder by a study of serial sections and by gross dissections in stillborn infants and in adults. These nerves course to the bladder in the endopelvic fascia and maintain a close relationship to the pudendal venous plexus. It is believed that these nerves are parasympathetic in function and that they aid, and possibly take over completely, the innervation of the bladder after certain types of pelvic surgery (for example, excision of the rectum) which interrupt the parasympathetic fibres passing through the pelvic plexus. It is also believed that the temporary atonic state of the bladder after these operations is due to traction on or trauma to these nerve fibres coursing along the perivascular route, but that they regain their function after a few days. Thus, if infection or over-distension of the bladder has been avoided during this phase of nerve shock, there will be a return of normal control of the bladder.

Nerve Grafts into Hands.

H. C. MARBLE AND C. B. BURBANK (*Am. J. Surg.*, March, 1953) discuss the use of free nerve grafts in reconstructive hand surgery. These grafts are indicated if it is impossible by any other method to perform either primary or secondary suture and if return of sensation to the hand is required. The authors have not had any patient who has had return of motor power; but sensation has returned, so that the patients could tell the difference between hot and cold and they knew when they were touching objects. While the sensation that returns is not normal, it is sufficient to increase the use of the hand. The nerve graft should not exceed one millimetre in diameter, but several strands should be used to fill a gap in a large nerve. Early nourishment is taken directly from the tissues, and this will maintain the outer layers only of a large graft. Homologous grafts are not effective in humans, although they may be in animals. The sural nerve is advised for the grafts, for the resulting loss of sensation does not often upset the patient. The ends of the host nerve are cut back till normal tissue is reached. Accurate end-to-end sutures of very fine silk or catgut are used to fix the graft, which should not be surrounded by any foreign material. Immobilization for three weeks is followed by gentle use and gentle physiotherapy. The success of the graft may be shown by abolishing all sensation by "Novocain" injection of the proximal host nerve.

Anastomosis of Right Bronchus to Trachea.

W. WEISEL AND R. J. JAKE (*Ann. Surg.*, February, 1953) report successful reimplantation of the right bronchus into the trachea following complete bronchial rupture due to an external injury forty-six days previously. The patient, aged thirty-two years, was involved in a car accident and suffered fractures of the right second to sixth ribs and of the left second to fourth ribs, right pneumohæmothorax, subcutaneous emphysema, fractured right acetabulum, and dislocation of the head of the right femur. After two days thoracotomy was performed for evacuation of blood and suture of lacerations of the right upper lobe. After another eighteen days he was very ill, and bronchoscopy, performed because of lack of expansion of the right lung, revealed a "bronchial obstruction". After a further twenty-six days his condition had improved sufficiently to permit further thoracotomy. Through the fourth right intercostal space the pleural cavity was opened, and a loculated empyema at the apex of the lung evacuated. The lung was found to be about six centimetres in diameter. Decortication of the lung was performed, and the hilar structures were displayed. The right pulmonary artery and veins were noted to be intact. The distal end of the right bronchus was found to be completely closed and retracted within the hilum of the lung. After removal of tenacious black mucoid material from the bronchus the lung was inflated with a syringe. A tracheal window was made in the right lateral wall above the carina, and then the distal bronchial stump was anastomosed to it with interrupted cotton sutures. An endotracheal tube was passed down into the right lung, which was then expanded by the anæsthetist. For the next three days this tube was aspirated half-hourly with a small catheter. Apart from a prophylactic tracheotomy on the fourth postoperative day (because of the fear of laryngeal œdema) convalescence was satisfactory. Sixty-four days after operation a bronchogram was normal apart from a minor distortion of the endobronchial pattern. There was no evidence of bronchiectasis. The patient returned to work eighty-seven days after the operation.

Carcinoma of the Thyroid Gland.

I. MACDONALD AND P. KOLIN (*Ann. Surg.*, February, 1953) have reviewed the literature on the place of surgery in the treatment of carcinoma of the thyroid gland and have reported six cases of their own. They state that there is now nearly unanimous acceptance of lateral cervical deposits of thyroid tissue as metastatic deposits of primary carcinoma of the thyroid gland, which may be clinically occult. Such spread may even be apparently confined to contralateral glands. In some cases multiple foci of intraglandular malignancy are present. From this review it is concluded that when a thyroid tumour is present with no clinical evidence of lymphadenopathy, the tumour with a reasonable margin of thyroid tissue should be excised. Then, if the lesion is a carcinoma, as determined by frozen or paraffin sections, extracapsular total thyroidectomy is performed with regional dissection of the lymphatic field from internal jugular to internal jugular vein. When there are evident lateral lymph-gland metastases, with or without an apparent lesion in the thyroid gland, biopsy of one of the glands is performed. If this demonstrates papillary carcinoma, radical block dissection is carried out on the involved side of the entire antero-lateral triangle *plus* the adjacent portion of the posterior triangle, especially in its lower third. The operation is completed by total extracapsular thyroidectomy and dissection of the glands on the opposite side as far out as the carotid sheath.

Common Duct Exploration for Stones.

FRANK GLENN (*Surg., Gynec. & Obst.*, October, 1952) reports 3439 operations for non-malignant disease of the biliary tract; 10·6% of the patients underwent choledochotomy at the time of the first operation and 2% underwent a second operation for choledochotomy. The mortality for the operation was 5·7%, compared with 0·5% for cholecystectomy alone. Of 100 patients followed carefully who had undergone cholecystectomy only at the first operation, seven underwent reexploration for symptoms suggesting stone in the common bile duct, and stone was found in four cases. In 100 cases of choledochotomy there was a similar recurrence rate. The author estimates that 10% of cases of cholelithiasis are associated with stone in the common bile duct. He lists the following indications for exploration at the first operation: (i) palpable stone in the duct, (ii) history of jaundice, (iii) thickened common duct wall with or without dilatation, (iv) contracted gall-bladder containing stones, (v) dilated cystic duct, (vi) enlarged head of the pancreas, (vii) patient of greater age than sixty years, with a prolonged history of biliary tract disease.

Thrombosis of Terminal Part of the Aorta.

FRANK V. THEIS (*Surg., Gynec. & Obst.*, October, 1952) reports 29 cases of complete occlusion of the lowest part of the abdominal aorta. The patients represented 1·6% of patients examined for peripheral vascular disease of the lower extremities. The average age of incidence was fifty-seven years, and sex incidence was equal. Hypercholesterolæmia was a common feature. In most cases the onset of symptoms was insidious, and most patients complained of intermittent claudication. Gangrene occurred in nine cases, in eight of which conservative surgery only was required. The author states that X-ray examination shows calcification of the aorta, and peripheral pulses are absent on both sides. Oscillometric examination shows a constant picture. Aortography is rarely necessary to confirm the diagnosis, but will reveal the site, extent and completeness of the occlusion. For treatment the author recommends bilateral lumbar sympathectomy and points out that resection of the terminal part of the aorta is hazardous and inadvisable.

Chronic Hydradenitis Suppurativa.

H. CONWAY *et alii* (*Surg., Gynec. & Obst.*, October, 1952) state that chronic *hydradenitis suppurativa* is chronic inflammatory disease of the apocrine glands, which are tubo-alveolar glands lying deep in the subcutaneous tissue in association with hair follicles and located in the axilla, breast, areola, groin, perineum and perianal and umbilical regions. Acne appears to predispose to hydradenitis, although poor hygiene, hyperhidrosis and use

of chemical depilatories may play a role. The clinical picture shows cutaneous nodules with inflammation and fibrosis and multiple abscesses with skin sinuses. In the early case cure may follow local incision with improved hygiene. In later cases excision of the area may be required with split-skin grafting.

Gastro-Jejunostomy.

J. M. ZUBIRAN *et alii* (*Arch. Surg.*, August, 1952) describe a series of experiments on dogs in which gastric secretion was studied in relation to gastric pouches of Heidenhain and Pavlov types. Analyses were made to show the effects of gastro-enterostomy. It produced a sustained increase in gastric secretion of from 31% to 140% of the original level. This rise occurred only when the anastomotic stoma was placed high in the body or fundus of the stomach, and not at all if the stoma was placed in the antrum near the pylorus. That this rise was due to the humoral factor in gastric secretion was shown by its occurrence in vagus-denervated Heidenhain pouches, and its prevention by excision of the pyloric antrum. The authors suggest that high gastro-enterostomy is much more likely to be followed by gastro-jejunal ulceration than similar anastomosis made low in the stomach.

Caecostomy and Colostomy in Colonic Obstruction.

J. H. ALBERS AND L. L. SMITH (*Surg., Gynec. & Obst.*, October, 1952) discuss the role of caecostomy and colostomy as emergency measures in 146 cases of large-bowel obstruction. They point out that the majority of these obstructions are of the closed-loop type owing to competence of the ileo-caecal valve. Perforation of the caecum occurred in 10 cases of the series. The authors emphasize the need for laparotomy at the time of the emergency operation and describe three cases in which inadequate surgery was performed because of a limited approach and failure to realize the full pathological picture. They found that tube caecostomy was inadequate for bowel decompression, and that in most cases the operation of choice was transverse colostomy.

Fascial Grafts for Repair of Inguinal Hernia.

MAURICE LEE (*J. Internat. Coll. Surgeons*, March, 1953) advocates the use of strips of *fascia lata* in the repair of inguinal hernia. He states that Gallie and Le Mesurier, of Toronto, introduced this method in 1921 because of the high incidence of recurrence after operation at that time. While the rate of recurrence may not be as high now as it was then, it is still the bane of every herniotomist. The use of strips of *fascia lata* has of recent years become less common, and the author maintains that in certain types of hernia it is questionable whether repair by silk or other suturing will prevent recurrence. *Fascia lata* in strips produces a wall of fibrous tissue which takes the place of a deficient or atrophied posterior wall of the canal. The author uses fascial grafts in hernias of six classifications: (i) All recurrent hernias. (ii) Large oblique inguinal hernias with a large defect in the *fascia transversalis*. If the hole in the *fascia transversalis* admits two or more fingers, the opening must be closed with stout catgut, just enough room being left for the emergence of the cord. This area must then be reinforced by a ring of *fascia lata*. (iii) All direct hernias. No true sac is present, and these hernias occur usually in elderly patients who have, in addition, weak abdominal muscles. (iv) Inguinal hernias in obese patients. The pot-bellied patient with muscular wall of the abdomen of poor quality and infiltrated with fat is a poor subject for the use of silk *et cetera*. (v) Sliding hernias. (vi) Hernias in patients with chronic bronchitis and cough. The author describes the incision he uses and the technique of the use of the fasciotome and the repair. He emphasizes the importance of anchoring the graft to the pubic tubercle by putting a bite into the periosteum. He states that only meticulous attention to detail by the surgeon will prevent recurrences.

Tracheo-Œsophageal Fistula.

N. L. LEVEN *et alii* (*Ann. Surg., October, 1952*) review a series of 103 cases of tracheo-œsophageal fistula. In 23 cases multiple-stage operation was performed with 13 survivals. In 68 cases primary œsophageal anastomosis was performed; 43 patients are living. After rehydration of the patient, the operation is performed through an extrapleural approach with resection of the right fourth rib. The azygos vein is divided, and the upper and lower ends of the œsophagus are mobilized and anastomosed. Post-operatively the baby is maintained on intravenous fluid therapy with vitamins and antibiotics. Twenty-four hours later gastrostomy is performed, and feeding is commenced by this route twelve hours later. Oral feeds are commenced on the tenth day, after the continuity and patency of the œsophagus have been checked with a small amount of lipiodol; if there is a leak at the anastomosis, oral feedings are withheld until this closes.

Vagotomy and Gastro-Enterostomy for Duodenal Ulcer.

CHESTER C. GUY, CARL Y. WERELIUS AND DONALD H. DEXTER (*J. Internat. Coll. Surgeons*, March, 1953) state that they favour, as a general routine, the use of vagotomy and gastro-enterostomy in the surgical treatment of duodenal ulcers. They were able to maintain a good follow-up of 170 patients on whom these procedures had been performed for duodenal ulcer from six months to five years previously. In 153 cases (90%) the result was satisfactory, in 17 (10%) it was fair or unsatisfactory. Two deaths occurred in a series of 201 cases. The authors give some detail of their technique and post-operative care; in discussing the relative merits of vagotomy with gastro-enterostomy and gastric resection they consider two major points—the technical details and the end results. They state that vagotomy with gastro-enterostomy requires about ninety minutes to perform, carries a mortality of 1% or less, and leaves the entire stomach on which additional surgical procedures can be carried out, if necessary, at a later date. Gastric resection requires two to three hours, has at least twice the mortality rate and involves removal of at least three-fourths of the stomach to obtain good results. If it is a failure, further surgical treatment usually is not advised. The authors state that while these details are highly important, they have less significance than the end results; the details of these are given.

The Fallibility of Skiagrams of the Colon.

J. M. T. FINNEY AND D. H. STONE (*Ann. Surg.*, May, 1953) have studied the reasons for the failure of early malignant lesions to be revealed on barium enema examinations, and emphasize the fact that all laboratory and allied procedures are rightly catalogued as diagnostic aids only. Mistaking a neoplasm for an inflammatory lesion, or vice versa, is not necessarily such a serious matter as the failure to find the neoplasm at all. Rectal lesions are often difficult for the radiologist to demonstrate, but such lesions are within the reach of the finger or the sigmoidoscope. The neoplasm of the colon is most often overlooked if the bowel is bound down by adhesions from a previous operation or inflammation, or if it is situated posteriorly in a relatively fixed caecum. The difficulty in diagnosis is illustrated by three cases which are quoted and in which operation was carried out despite the normal radiological findings.

Arteriography.

I. MACKENZIE AND H. A. R. HAMILTON (*Brit. J. Surg.*, March, 1953) discuss the technique of arteriography and its use in the diagnosis of peripheral arterial lesions. They state that light general anæsthesia is used, chiefly to ensure complete immobility of the limb during the taking of the film. Then, with the patient supine, the hip is fully externally rotated and partly abducted and flexed while the knee is flexed to a right angle or rather less. The casette is rested on a sandbag under the leg. After the artery is displayed 15 or 16 mils of "Pyelosil 70" are injected into the common femoral artery through a number 18 or 19 serum needle which is directed proximally. The injection is given at the rate of 1·5 mils per minute, and then an exposure is made at the end of the injection. After withdrawal of the needle any oozing from the puncture hole can be controlled by gentle pressure with a swab.

Carcinoma of the Pancreas.

E. E. CLIFTON (*Arch. Surg.*, August, 1952) reports an investigation regarding clinical picture and treatment in a series of 122 cases of carcinoma of the pancreas. Pain was the most frequent symptom; anorexia with loss of weight and jaundice were symptoms of shorter duration. Half the patients had liver enlargement, but only 22% had clinical enlargement of the gall-bladder. Radiological examination showed positive findings in only 8% of those examined; 73% had surgical intervention, 10% undergoing resection of the pancreas and duodenum. The operative mortality for palliative surgery was 38% compared with 33·3% for resections. The average survival times were 5·4 months with palliative treatment and 2·5 years with resection.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LXXXI.

DIAGNOSIS AND TREATMENT OF CONGENITAL DEFECTS OF THE HEART.

IN 1938 Gross successfully ligated a patent *ductus arteriosus* for the first time. Blalock devised and carried out his operation for the relief of patients suffering from Fallot's tetralogy in 1944, and in 1945 Crafoord and Gross independently described successful operations for coarctation of the aorta. Stimulated by these achievements, cardiologists all over the world set to work to unravel the intricacies of congenital cardiac defects, and as a result of these labours, Paul Wood was able to say in 1950 that "modern techniques have made it possible to prove the precise nature and physiological effects of 90% of all congenital cardiac anomalies during life". Despite this, selection of patients for operation may tax the resources of a fully equipped cardiological unit. In any article of this type it is impossible and undesirable to consider fine detail, but understanding of a few major principles should enable the average doctor to make a reasonable diagnosis in a majority of the cases.

The most important congenital heart defects are abnormal openings or stenoses or combinations of these.

Openings may be between the right and left ventricle, the right and left auricles or the aorta and the pulmonary artery. Stenoses may be in the valves or the chambers of the heart, or in the large vessels leaving the heart.

Pure stenoses of surgical significance are coarctation of the aorta and pure pulmonary stenosis. Central cyanosis does not occur with these defects, because, however much the circulation may be impeded, all the blood that reaches the aorta must pass through the lungs. Peripheral cyanosis such as is seen with any circulatory failure may occur in terminal phases.

Pure openings or shunts are patent *ductus arteriosus*, atrial septal defects and ventricular septal defects. Because of the high pressure in the left or arterial side of the heart, flow is from left to right through these openings, and so again there is no cyanosis. The shunted blood returns to the left side of the heart through the lungs, adding to the pulmonary blood flow, and if the shunt is of more than slight degree the pulmonary vascular markings will be increased; as a corollary, heavy vascular markings in the peripheral lung fields always mean a large left-to-right shunt.

If there is an atrial septal defect or a ventricular septal defect and in addition an obstruction to the outflow from the right ventricle, cyanosis occurs because the pressure in the right side of the heart is raised and blood is shunted from right to left. Central cyanosis nearly always means an intracardiac shunt *plus* obstruction to the outflow from the right ventricle. Exceptions to this are transposition of the great vessels and abnormalities of the venous return to the heart. In the Eisenmenger syndrome, pulmonary hypertension, and not pulmonary stenosis, is associated with reversal of flow through a ventricular septal defect, or less often an atrial septal defect or persistent *ductus arteriosus*.

Morbus Cæruleus.

The two conditions of surgical importance in the cyanotic group are, first, Fallot's tetralogy and, second, pulmonary stenosis with a patent *foramen ovale* or a larger atrial septal defect, but with an intact interventricular septum. In Fallot's tetralogy there are pulmonary stenosis and a ventricular septal defect with a shift to the right of the aorta, so that it overrides both ventricles, and a large right ventricle. In both these conditions blood-flow through the lungs is greatly diminished, so that the peripheral lung fields are unusually clear. Pulmonary plethora with obvious cyanosis is very suggestive of transposition of the great vessels. It may be found, too, in the Eisenmenger syndrome, usually with less marked or inconstant cyanosis, when a left-right shunt has undergone reversal of flow due to pulmonary hypertension. However, when the syndrome has been present from birth the peripheral lung fields may be clear, although the main pulmonary artery is dilated.

In order that we may assess the vascular markings in the lung fields, films must be taken with a standard degree of expansion of the lungs; and as it is difficult to control the breathing of young children, it may be extremely hard to make an assessment in those under three years of age. Many of the babies with severe and hopeless defects die before the age of three years, and after this age approximately 70% of all "blue babies" are suffering from Fallot's tetralogy.

The characteristics of Fallot's tetralogy are as follows:

1. Central cyanosis of moderate or great severity present from birth or early infancy.

2. Clubbing of fingers and toes.

3. A history of squatting.

4. Small pulmonary arteries with diminished density of lung fields.

5. Right ventricular preponderance in the electrocardiogram.

6. Absence of marked cardiac enlargement despite hypertrophy of the right ventricle (a cardio-thoracic ratio not greater than 54%).

7. A systolic murmur maximal in the second or third intercostal space close to the sternum, and a palpable thrill in half of the patients. The second heart sound is invariably single. With complete atresia there may be no murmur, or a low continuous murmur due to anastomotic vessels may be heard.

A similar picture with left ventricular preponderance and a large left ventricle suggests tricuspid atresia.

Pulmonary stenosis with an atrial septal defect and not a ventricular septal defect shows a clinical picture very similar to that of Fallot's tetralogy, but with significant differences. Cyanosis may appear later in these patients. The heart is larger than in Fallot's tetralogy, and the main pulmonary artery is usually dilated. A systolic murmur and thrill are always present in the pulmonary area, and the second heart sound is always single. When there is doubt about the clinical diagnosis, cardiac catheterization or angiocardiography may be used.

Cardiac Catheterization.—A plastic radio-opaque catheter is passed into a vein at the elbow and pushed on until it enters the heart. Its passage is controlled on the X-ray screen, and information is obtained by watching the path of the catheter, by taking pressure readings from various chambers of the heart and the pulmonary artery, and by estimating the oxygen content of the blood taken from different situations. The catheter can be passed into the right auricle and on into the right ventricle or down the inferior *vena cava*. From the right ventricle it can be passed into the pulmonary artery, but unless there is a septal defect through which the catheter may pass, it is impossible to enter the left side of the heart. Cardiac catheterization is particularly helpful in demonstrating left to right shunts and in proving pulmonary stenosis. It is therefore most helpful in acyanotic cases.

Angiocardiography.—Angiocardiography on the other hand is most useful in demonstrating right to left shunts and is most helpful in cyanotic cases. A 70% solution of "Diodone" or similar dye is injected as quickly as possible into a vein at the elbow, and a series of eight or more films is taken in ten seconds. Normally the right auricle fills in one second. The right ventricle and pulmonary artery fill in two seconds. The pulmonary veins and the left auricle fill in four to five seconds, and the left ventricle and aorta fill in six to eight seconds. If there is a free right-to-left shunt, the aorta fills at the same time as the pulmonary artery. Angiography may also show details of cardiac and arterial anatomy useful in deciding the approach for operation. Angiography is not without risk in severely distressed patients, and sometimes, especially in young children whose outlook without operation is hopeless, it is preferable to perform an exploratory thoracotomy. In any case operations for "blue babies" should be avoided under the age of three years unless survival beyond this age seems unlikely.

The object of treatment in surgical relief of "blue babies" is to increase the volume of blood flowing through the lungs, and this can be achieved in two ways. By joining a systemic artery to a pulmonary artery an artificial *ductus arteriosus* is created, and a considerable amount of blood may be diverted from the aorta into the pulmonary artery in this way. Blalock's operation effects this principle by anastomosing a subclavian artery to a pulmonary artery, and in the Willis Potts operation a direct opening is made

between the aorta and the pulmonary artery. The same result may be achieved by enlarging the stenosed pulmonary orifice, as originally attempted by Doyen in 1913 and reintroduced by Brock in 1948.

Pulmonary stenosis may be valvular when the fused cusps of the pulmonary valve form a dome-like structure with a tiny central orifice, projecting into the pulmonary artery, which is always dilated distal to the fused valve, or infundibular when the narrowing is in the outflow tract of the right ventricle proximal to the pulmonary valve. With infundibular stenosis the pulmonary artery is not dilated.

Brock's operation is very satisfactory when the stenosis is purely valvular, but less so in infundibular stenosis.

When there is pulmonary stenosis without a ventricular septal defect, relief can be obtained only by the direct operation; fortunately nearly all these patients have valvular and not infundibular stenoses. When there is no intraventricular septal defect, the right ventricle can empty only through the constricted pulmonary orifice, and the presence of an artificial ductus by raising the pressure in the pulmonary artery makes emptying of the right ventricle more difficult and hastens its failure.

When there is a ventricular septal defect, as in Fallot's tetralogy, either type of operation will give an immediate good result; but on physiological grounds, Brock's operation is probably better, provided that an adequate and lasting correction is achieved. Most surgeons now prefer it when the stenosis is purely valvular, but few have yet adopted it for infundibular stenosis.

Patent Ductus Arteriosus.

A machinery murmur in the pulmonary area in a patient who is acyanotic should suggest a patent *ductus arteriosus*. The diastolic element in the murmur is best heard if the breath is held in full expiration. It may not be recognizable in very young children. The child may be normally developed and active, or puny and undersized. The pulse pressure is usually increased because of the low diastolic pressure, but this is not always noticeable. X-ray examination shows a slightly enlarged heart and a dilated pulmonary conus, and the vascular markings in the lung fields are increased. Screening examination confirms these findings and may show pulsation in the pulmonary vessels. Electrocardiography usually shows little abnormality.

The flow of blood is from the high-pressure aorta to the low-pressure pulmonary artery; but in a small proportion of patients who develop pulmonary hypertension, the pressure in the two vessels becomes approximated, and flow may cease and the characteristic murmur may disappear. In a small but important group with some degree of pulmonary hypertension, a systolic murmur in the pulmonary area, congested lung fields, prominent pulmonary artery, and signs of right as well as left ventricular hypertrophy in the electrocardiogram, may suggest the diagnosis of ventricular septal defect, and in a few with severe pulmonary hypertension a reversed shunt may result in cyanosis. In these cases cardiac catheterization is required to make a diagnosis.

The risk of operation on patients with uncomplicated patent *ductus arteriosus* is between 1% and 2%, and operation should always be advised as soon as the diagnosis is certain, in children and young adults.

In older people who show no evidence of cardiac strain, operation may perhaps be deferred, but it should not be delayed if the heart is enlarging or endocarditis has occurred. Endocarditis usually clears up after operation; but now that it can be controlled by chemotherapy, intensive treatment with suitable drugs should always precede operation.

Coarctation of the Aorta.

Coarctation of the aorta is very easily recognized in the great majority of cases and should be suspected in any young person with a raised blood pressure. An absent or feeble and delayed femoral pulse confirms the diagnosis. Visible or palpable pulsating arteries around the angle of the scapula and on the chest wall below the axillæ are usually easily detected. X-ray examination of the chest shows notching of the ribs in most patients, but below the age of seven or eight years notching may be absent.

The blood pressure in the femoral arteries, if it can be determined, will be below that in the arms. Normally, as determined by the cuff method, it should be 10 to 20 millimetres of mercury higher. Usually there is a systolic murmur at the base of the heart, and if there is an associated patent *ductus arteriosus* or other defect more complicated murmurs may be present.

Every patient with coarctation should be considered with a view to operation, because the complications of coarctation are frequent and severe, and although some patients live normal lives and die of old age, the majority die under forty years of age. The risk of operation at the present time is less than 10%. Probably the ideal time for operation is between the ages of eight and twelve years. Before the age of eight years, operation is indicated if there is evidence of increasing heart strain, or if the child is puny or under-developed. In these children the aorta is small, and it is not yet certain that the anastomosis always grows with the aorta. After the age of eight years, the aorta is usually large enough to provide a sufficient flow for full adult activity, even if the anastomosis has not grown. Operation is much easier and safer in children than in adults, and after the age of twenty years atheromatous changes in the aorta tend to increase the difficulty and risks of operation.

Excision of the stricture with end-to-end anastomosis of the divided aorta is the treatment of choice for these patients. This may be impossible because of the length of the stricture or because the aorta in older subjects is inelastic or because the presence of a complicating aneurysm increases the length of the aorta that has to be resected. For these patients Gross has introduced the method of using a graft of preserved aorta, with considerable success. Grafts should not be used in children, because it is certain that the graft will not enlarge as the aorta grows.

As an alternative, the subclavian artery may be divided and turned down and sutured to the distal end of the aorta; but although excellent results may be obtained by this method, it is less satisfactory than excision and direct anastomosis.

Congenital Vascular Rings.

Congenital vascular rings result from abnormal development of the great vessels. There may be a double aortic arch with one limb in front and the other behind the œsophagus and trachea, or a right arch may pass behind these structures and the ring be completed by a patent or obliterated ductus tying the posterior part of the aorta to the anterior pulmonary arteries. There are many variants, and the exact anatomy can rarely be determined until the chest is opened.

Symptoms may appear in infancy or not until later life when the great vessels are dilating and hardening. Typically an infant presents with a wheeze and a history of frequent respiratory infections and often with difficulty in swallowing or regurgitation of food. A barium meal examination shows indentation of the œsophagus, and bronchoscopy and bronchograms may help. All these investigations are difficult in infants. In older patients difficulty in swallowing is the usual complaint, because the œsophagus is more readily compressed than the adult trachea.

Operative division of the ring is usually possible and is the only way of relieving these patients.

Simple Pulmonary Stenosis with Closed Septa.

Cyanosis has often been looked on as a symptom of pulmonary stenosis, but this is true only when there is a septal defect to allow a right-to-left shunt. With closed septa central cyanosis cannot occur, but in severe cases there may be some blueness of the extremities due to capillary stagnation. There is a systolic murmur and thrill high in the pulmonary area. The X-ray film always shows some enlargement of the main pulmonary artery, and right ventricular enlargement, varying with the severity of the condition, will be shown by the clinical signs, X-ray appearances and electrocardiogram. Cardiac catheterization and/or angiocardiography will usually be needed for final assessment; and if the symptoms justify interference, pulmonary valvulotomy (Brock) gives gratifying results.

Ventricular Septal Defect.

Despite some brave attempts, no satisfactory operation has yet been devised for the repair of a ventricular septal defect, but the condition is important in differential diagnosis. Mild cases show little except a harsh systolic murmur and thrill (Roger's murmur) in the third and fourth left intercostal space close to the sternum; but with large defects there is increasing enlargement of the heart with plethora of the lung fields and a large pulmonary artery, and diagnosis from patent *ductus arteriosus* with

pulmonary hypertension may require cardiac catheterization. Atrial septal defect may cause confusion in some cases.

Atrial Septal Defect.

Patients with an atrial septal defect have a systolic murmur in the pulmonary area, and may have a thrill; and in some cases a diastolic murmur is also heard in the pulmonary or mitral areas. The second heart sound is split in practically all cases.

Electrocardiography shows right ventricular preponderance, and frequently there is some evidence of right bundle branch block.

X-ray examination shows right ventricular enlargement, dilatation of the pulmonary artery and pulmonary plethora, and screening examination may show a well-marked hilar dance. With a small defect disability is often minimal, but large defects may be crippling.

Many attempts have been made to close these defects surgically. Ideally, this could be done through an opening in the right atrium with a quiet, empty heart while the circulation and respiration were maintained by an artificial heart-lung machine. Much progress has been made in the development of these machines, and one or two attempts have been made to use them for this purpose; before long it seems likely that they will be in regular use.

The flow of blood through the heart can be stopped by occluding both *venæ cavæ* and/or the aorta and pulmonary artery, and this can be tolerated in normal people for about two and a half minutes. If the patient is refrigerated to lower metabolism, circulation can be stopped for periods up to ten or twelve minutes, and Bailey and others have used this method to close a few atrial septal defects with some success.

Gross has developed the most ingenious and practical method yet devised. Recognizing that the pressure in the right atrium even with a septal defect is only a few centimetres of water, he has constructed a "Latex" rubber funnel about 10 centimetres high, and after placing a clamp across the right atrium makes an incision in its wall and sews the lower end of his funnel to its edges. When the clamp is removed, blood rises in the funnel for a few centimetres, and through the pool of blood the surgeon can explore the defect and close it by direct suture, or by the application of a patch of "Nylon" or polythene. Gross has used this method in a number of desperately sick patients, and in his first seven cases has had three striking successes; it seems probable that this method will prove of real value.

C. J. OFFICER BROWN,
Melbourne.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Melbourne, on May 13, 1953.

Displaced Lower Femoral Epiphysis Occurring at Birth.

DR. ELIZABETH MCCOMAS described the case of an infant, six and a half weeks premature, with a birth weight of four pounds six ounces, who presented at birth by the breech with extended legs. Fourteen hours later she was admitted to the Queen Victoria Hospital, and the accompanying letter from the practitioner suggested possible damage to the right femur.

A skiagram taken soon after admission revealed that the epiphysis of the lower end of the left femur was displaced anteriorly and proximally—the epiphysis was lying in front of the lower end and a small flake of bone had been avulsed from the shaft.

One week after the child's admission to hospital manual reduction of the displacement was attempted but failed, so on the following day an open reduction was performed. The epiphysis had become attached to the anterior surface of the femur and had to be prised off before reduction could be achieved. Stability could be obtained only with the knee flexed, so the leg was immobilized in a plaster of Paris cast in that position.

A skiagram one day later showed that the epiphysis of the lower end of the left femur was approximately in the normal position. The bone flake was restored to its correct place. On the right side, subperiosteal calcification was evident at the lower end of the femoral shaft.

The plaster of Paris cast was removed three weeks later and radiological examination revealed a massive callus at the lower end of the shaft of the left femur. The epiphysis appeared normal and had grown a good deal since the injury. The femoral shafts were equal in length.

One month later, when reviewed in the out-patient department, the infant was clinically normal. Radiological examination showed that the subperiosteal calcification had been almost entirely absorbed, and the position and development of the epiphysis were normal.

After the clinical presentation of the case Dr. McComas demonstrated the relevant skiagrams.

DR. J. B. COLQUHOUN said that he had not met with a similar case in the newborn. The obstetrician had shown considerable acumen in suspecting an injury even though he thought that it was on the opposite side. Dr. Colquhoun pointed out that epiphyseolysis of the lower end of the femur was once a form of treatment for *genu valgum*; sometimes part of the metaphysis was also displaced in such a procedure, with resulting deformity, and the method was not favoured now. Dr. Colquhoun said that the management of the infant was good. He was surprised that manipulation had failed, and that an osteotome had been necessary to separate the epiphysis from the metaphysis.

DR. E. E. PRICE said that he had met with only one similar case, in which treatment was necessary to prevent angular deformity of the lower end of the femur.

Panner's Disease.

DR. W. R. FORSTER briefly reviewed the condition of osteochondritis of the capitulum of the humerus. He said that the condition had first been described by Panner, of Copenhagen, in 1927, and was a relatively unknown disease. A search through the literature disclosed only 13 reported cases; but if in every suspected case the patient was examined radiologically, some of the conditions which were labelled "joint strain", "tennis elbow", and "synovitis" might be found to be cases of Panner's disease. It appeared to be more frequent in boys than in girls and frequently followed mild joint injury. The described cases had occurred in patients between the ages of seven and ten years.

The condition appeared to be closely allied to disorders in other epiphyses, mostly known by the name of the surgeon who first described them—such as Legg-Perthes-Calvé's disease of the hip, Köhler's disease of the scaphoid bone, and Osgood-Schlatter's disease of the tibial tuberosity. No epiphysis was immune from this strange disorder. As the suffix "itis" suggested an inflammation, perhaps the various disorders could be more accurately described as "osteochondro-dysplasias" or even less specifically as localized rarefying conditions of bone.

Dr. Forster then described the case of a boy, aged eight years, who had presented at the Children's Hospital with the symptoms of tenderness on the lateral condyle of the humerus just above the head of the radius, and pain in the region of the elbow joint, made worse by attempts to increase the degree of flexion or extension, the full range of which was slightly limited. The child had no increase of body temperature, and there was no muscle wasting in the region of the elbow. The tuberculin sensitivity test result was negative. There was a history of a fall with twisting of the elbow joint a few weeks previously.

Dr. Forster, before describing the radiological features of the case, demonstrated skiagrams of elbow joints of several normal children of varying ages. The development of centres of ossification according to age was shown. Not until after the age of ten years were all the ossific centres visible radiologically.

In contrast to the skiagrams of normal children, those of the patient showed several abnormalities. The capitulum on the affected side was smaller in size than that on the normal side. There was an absence of cancellous tissue on the diseased side, and in the epiphysis there were areas of osteoporosis and areas of osteosclerosis giving the appearance of fragmentation and irregularity in shape. The joint space appeared to be wider than on the normal side. Dr. Forster said that the radiological description could just as easily apply to a case of Perthes's disease.

The treatment of the patient was simple immobilization of the joint by means of a plaster of Paris cast, with the elbow flexed to a right angle. Progress was followed by means of serial skiagrams. At the time of presentation there was neither pain nor limitation of movement. Simple immobilization was sufficient, as the joint was non-weight-bearing.

DR. J. B. COLQUHOUN, in opening the discussion, said that the condition was by no means as common in the humerus as in the upper part of the femur, probably because of the absence of weight-bearing in the former. The frequency of loose bodies in the elbow joint might be due to unrecognized osteochondritis. A similar affection of the elbow region was *osteochondritis dissecans*, or "quiet necrosis of the capitulum", first described by Paget, and also responsible for some cases of loose bodies in the elbow joint.

Dr. Colquhoun said that he had seen some patients whose radiological features closely resembled the early skiagrams of Dr. Forster's patient. Subsequent examinations revealed a spontaneous return to normal, presumably because there was no weight-bearing.

It appeared that trauma was an important factor in the ætiology of the disease, with interference in blood supply leading to avascular necrosis of bone with decalcification and softening. Later, the reverse process with recalcification and return to a normal state might occur.

Treatment consisted of immobilization with prevention of weight-bearing or further trauma. There was no indication for surgical intervention.

DR. COLIN MACDOALD said that he was convinced that the condition represented a disorder of osteochondral development. It was doubtful whether trauma with avascular necrosis of bone was an important factor. The condition should not be confused with *osteochondritis dissecans*.

DR. E. E. PRICE said that he, too, thought that the condition was due to avascular necrosis and that the juvenile and adult forms of osteochondritis as well as *osteochondritis dissecans* were essentially due to the same underlying cause, namely, interference with blood supply.

Dr. Price then described the blood supply to epiphyses, and demonstrated how a compressive force would distort and constrict blood vessels in the Haversian systems, leading to an avascular state and rarefaction, which would ultimately result in epiphyseal condensation, commencing at the centre but encroaching on the periphery. Similar disorders were responsible for osteochondritis in the region of the hip and knee joints. When the disorder was arrested by immobilization or freedom from weight-bearing, the affected zone was replaced by new bone as a result of osteoblastic activity commencing at the periphery. The process of healing was comparable with that occurring after bone-grafting.

Congenital Ovarian Agenesis.

DR. ELIZABETH K. TURNER described a case of probable congenital ovarian agenesis in a child twelve months of age, with associated abnormalities of *pterygium collis* and coarctation of the aorta. The case will be described in detail at a later date.

Congenital Heart Block Diagnosed Before Birth.

DR. K. J. RENNIE said that the case to be described was one in which congenital heart block was diagnosed in the ante-natal period, thirty-six hours before birth. He pointed out that congenital complete heart block was a well-known condition, but few cases of congenital partial heart block had been reported. Obstetrically the cases were of interest, as an erroneous diagnosis of fœtal distress might be made and induction of labour or even Cæsarean section performed.

The patient, a third *multigravida*, aged thirty-three years, was admitted to Queen Victoria Memorial Hospital in September, 1952, at full term. Her previous two pregnancies had been normal, and she had been quite well during the latest pregnancy apart from having had a febrile "cold" six weeks prior to admission to hospital. On examination of the patient, the fundus was at term and the vertex was presenting and unfixed. The fœtal heart could be clearly heard, but it was completely irregular, the rate being 120 beats per minute, and the heart sounds were quite unlike the characteristic muffled sounds associated with fœtal distress. A vaginal examination was made to exclude a cord presentation, and no abnormality was found. The membranes were intact. A provisional diagnosis of fœtal multiple extrasystoles was then made, and it was decided to allow labour to proceed normally. On the following morning the rhythm of the fœtal heart was found to be a succession of three beats followed by a pause, and congenital incomplete heart block was then suspected.

The patient was delivered that night after a normal labour. The baby, a female weighing seven pounds fifteen ounces, cried normally at birth and was a good colour. On examination, the heart was found to be in the normal position and not enlarged; the rhythm was completely irregular, the rate being 120 beats per minute. No murmur was heard. Electrocardiography was attempted, but was unsuccessful owing to technical difficulties. The baby was discharged from hospital on the seventh day after birth, when she appeared to be thriving. There was no change in the heart rate or rhythm.

The baby was next seen in the out-patient department at the age of ten weeks. There had been no cyanotic attacks, and progress was normal. The heart sounds were still very irregular and almost inaudible. No murmur was heard. An electrocardiogram showed a varying degree of partial heart block—mainly alternating 2:1 and 3:1 ratios—but other portions of the tracing, and also an electrocardiogram taken at a later date, had the appearances of complete heart block. The heart rate was 68 beats per minute. The *P-R* interval varied from 0·18 to 0·26 second. A skiagram and screening examination of the chest with a barium bolus showed no abnormality.

The baby was seen again at the age of five months, when she weighed sixteen pounds and was obviously thriving. She had had no attacks of cyanosis or of syncope. The heart was not enlarged, no murmur was heard, and the heart sounds were very slow, muffled and irregular. The heart rate was 38 beats per minute. The electrocardiogram was similar to the previous one, mainly showing a varying degree of partial heart block, but some sections of the tracing had the appearances of a complete heart block. The *P-R* interval varied from 0·20 to 0·38 second.

The most recent electrocardiogram, taken when the baby was aged eight months and weighed eighteen pounds, had the appearances of complete heart block, the *P-R* interval ranging from 0·08 to 0·34 second. Clinically the heart was not enlarged, the sounds were more easily heard than on previous occasions, and no murmur was heard. The baby was at present progressing normally, and there had been no cyanotic or syncopal attacks.

Dr. Rennie said that it had not been completely determined whether there was complete or only partial heart block in this case. Some sections of the electrocardiograms had the appearances of partial heart block with Wenckebach periods, whilst others showed complete heart block. The most recent electrocardiogram had the appearance of complete heart block.

Other interesting features of this case were the diagnosis of congenital heart block before birth, the failure to demonstrate any other signs of congenital heart disease, and the absence of any symptoms in the baby, such as cyanotic or syncopal attacks.

The most likely cause for the heart block in the present case appeared to be some congenital deficiency or deformity of the fibres in the bundle of His.

In conclusion, Dr. Rennie thanked Dr. Kate Mackay for interpreting the electrocardiograms.

DR. M. L. POWELL congratulated Dr. Rennie on the diagnosis of the disorder. He said that the youngest infant with heart block diagnosed at the Children's Hospital was just under one year of age, with a heart rate of 48 per minute. The prognosis appeared good, and except for occasional syncopal attacks on exertion, patients could live reasonably uneventful lives even with the persistence of complete heart block.

DR. JOHN COLEBATCH quoted a recent case at the Women's Hospital. The mother was hypertensive, fœtal heart irregularities were heard, and the heart rate was 124 per minute. There was a tendency to periodicity. A diagnosis of congenital heart block was made, but an electrocardiogram soon after birth demonstrated that the irregularity was due to extrasystoles. In the literature attempts to record electrocardiograms of the *fœtus in utero* had been reported.

DR. H. G. HILLER said that it was possible that the disorder might have been complete heart block from the start. Complete heart block usually manifested itself with an irregular rhythm in infants in contrast to the regular rhythm found in adults.

Dr. K. J. Rennie quoted another case in which the patient, when examined ten minutes after birth, was found to have every fourth beat dropped. An electrocardiogram had demonstrated a sino-auricular block. The rate became regular after eighteen hours and had remained so since.

Miliary Pulmonary Lesions of Uncertain Ætiology.

DR. M. J. ROBINSON presented the case history of a boy, aged twelve years and seven months, who had been referred to the Children's Hospital in 1943, at the age of two and a half years, with a history of vomiting, anæmia and failure to gain weight satisfactorily since the age of seven months. Before his admission to hospital, anæmia, which had not responded to iron or liver, had been treated by transfusion. At the Children's Hospital a diagnosis of cœliac disease had been made, and he had responded well to a diet of low fat

content. However, the hæmoglobin concentration had remained between 8·5 and 10·0 grammes *per centum* despite treatment with iron and liver extract. The child had been reviewed at intervals in the out-patient department, and his progress was considered satisfactory, although he suffered from fat intolerance with large offensive pale stools. By 1946 his hæmoglobin concentration was normal and he was very well.

In October, 1952, he was again referred to the Children's Hospital with a history of recurrent febrile "colds" since the age of three years. Tonsillectomy and removal of adenoids at the age of five years had made no improvement. His cough was intermittent and unproductive of sputum, but occasionally caused him to vomit. He had been an in-patient at a country hospital six months previously with pneumonia. Even at that stage he tolerated fats poorly and was referred with the diagnosis of fibrocystic disease of the pancreas. A recent skiagram of the chest suggested miliary tuberculosis. The family history gave no information apart from the fact that his father was a miner, and recent chest skiagrams of the family were reported to be clear. The only other information obtained was of occasional tightness in the chest and some shortness of breath on exertion.

On examination, he did not appear ill but was rather small for his years. His height was fifty-three and a half inches and he weighed four stone seven pounds. The only abnormal physical findings were a spleen palpable two fingers' breadth below the left costal margin and a mild degree of lymph gland enlargement involving the axillary and left supratrochlear glands.

Radiological examination of the chest showed a heavy miliary distribution in the basal zones. The lesion appeared much more diffuse and reticulated than miliary tuberculosis. Appearances were rather "hard" and suggested that the lesion had been present for a long time.

The following investigations, all of which yielded normal findings, were carried out: tuberculin sensitivity test with 1:1000 old tuberculin; microscopic examination of the urine; radiological examination of the hands; blood amino acid absorption curve; culture of sputum; microscopic examination of the fæces; full blood examination; Coombs test; assay of duodenal secretion for tryptic activity.

The child was discharged from hospital, the diagnosis of cystic fibrosis of the pancreas having been disproved, but no alternative diagnosis was suggested. The splenomegaly persisted, but the boy was quite well. Repeated investigations gave no further information, a provisional diagnosis of reticulosis was made, and the boy was admitted to hospital for lymph gland biopsy. The findings from a bone marrow biopsy were normal, as were the serum protein content and albumin-globulin ratio. The erythrocyte sedimentation rate was 30 millimetres in one hour. The pathologist made the following report on the lymph gland: "The normal lymph gland structure is not apparent. There is an increased number of lymphocytes present, with single or small groups of reticulum cells scattered amongst sheets of lymphocytes. The lymphocytes do not extend beyond the gland capsule. One of the glands shows an increased amount of fibrous tissue throughout its substance. I would think that the above changes are probably an unusual chronic non-specific inflammatory reaction." A cervical lymph gland was removed for culture for viruses and rickettsia but none were grown. Serum urea determination and histoplasma tests showed no abnormality; but although the results of agglutination tests for *Salmonella typhi*, *S. paratyphi* A, *S. paratyphi* B, and *Proteus* were negative, there was agglutination to *Brucella abortus* (Bang's method) between titres of 1:80 and 1:640. Culture of bone marrow and blood did not produce a growth of the organism, but at that stage the brucellin skin test gave a strongly positive result.

As the splenomegaly and a raised erythrocyte sedimentation rate persisted, he was given a course of 250 milligrammes of aureomycin six-hourly for fourteen days. When last seen he was afebrile and quite well, the erythrocyte sedimentation rate had fallen to eight millimetres in one hour, and he had gained a little weight. The radiological picture had not changed, and the spleen, although not as large as formerly, was still palpable.

Dr. Robinson concluded by asking if the case was one of brucellosis with miliary pulmonary lesions.

DR. JOHN PERRY demonstrated by microprojection the histopathological appearances of the lymph gland removed for biopsy. The lymph gland showed a chronic non-specific inflammatory reaction—a sinus reticulosis. Dr. Perry said that serial agglutination tests carried out by ordinary methods over a period of two months had shown an insufficient rise in titre to be unequivocal. However, Wilson's modification of Coombs and Hausman's technique showed a rise in titre from 1:320 to 1:640 over the same period. That test sometimes revealed cases of brucellosis when ordinary techniques failed, and might indicate developing infection. Dr. Perry said that there was no reason why the ubiquitous granuloma of brucellosis could not occur in the lungs.

DR. JOHN COLEBATCH said that brucellosis was an uncommon infection in Australia and its incidence in children appeared to be even lower than in adults. The organism cultured in reported cases in Australia had always been *Brucella abortus*, except in a few patients who had come from overseas. It was therefore illuminating to learn that calves were immune to infection (Cotton), and that in Mediterranean countries, where brucellosis was prevalent, children showed a greater natural immunity to infection with *Br. abortus* than to infection with *Br. melitensis*. In the Melbourne Children's Hospital during the past nine years only six cases of brucellosis had been diagnosed, though it was possible that some mild cases had passed unrecognized. The diagnosis of brucellosis was made very difficult by the protean and usually unimpressive character of its manifestations. Brennemann had stated that it was the rule, not the exception, for the patients to have no signs at all. A presumptive clinical diagnosis might sometimes be made in the presence of one of the following clinical pictures: (i) pyrexia of unknown origin; (ii) a mild acute infection with weakness, sweats, chills and vague aches and pains; (iii) a more severe illness with enlargement of the spleen and the lymph glands, and pain over the spine; (iv) a chronic illness characterized particularly by weakness with fever, sweats, backache, headache, insomnia and mental depression. However, a number of cases occurred without having presented in any of those relatively characteristic ways. No matter how suggestive the clinical picture, therefore, no diagnosis could be established without the aid of the pathologist.

Of the various laboratory procedures available, the test for agglutinins in the serum was the most valuable, as a rapid screening method at least. Histopathological examination of the bone marrow, a lymph gland or other tissues would sometimes provide strongly suggestive or supporting evidence. Examination of the blood commonly showed neutropenia or lymphocytosis with some atypical lymphocytes. The intradermal brucellin test would give evidence of past contact with brucella infection, analogous to the evidence provided by the Mantoux test in tuberculosis. Culture of the organism, however, from the blood or elsewhere, provided the only definite proof of a diagnosis of brucellosis.

Dr. Colebatch then briefly traced the histories in the six cases that had been diagnosed in the Children's Hospital. The provisional diagnosis on the patient's admission to hospital was rheumatic fever in two cases, arthritis of the hip in one case, splenomegaly with fever in one case, and pyrexia of unknown origin in one case. The result of the agglutination test was positive in all cases, the titres ranging from 1:240 to 1:4096. In no case was a positive blood culture result obtained.

The case presented by Dr. Robinson had presented four points of interest, which Dr. Colebatch discussed. The first problem had been that of diagnosis in the case of a pale thin boy with a palpable spleen, radiological signs of a miliary lesion in the lungs, a consistently raised blood sedimentation rate and negative findings from numerous other investigations. It was the history of recurrent pyrexia in 1952, even though associated with "bronchitis", which had prompted the request for the brucella agglutination test and for the skin test. Although those tests gave positive results, Dr. Colebatch asked whether one could definitely accept the diagnosis of brucellosis in the present case, without having grown the organism.

The second problem concerned the radiological changes, which Dr. Hiller had astutely recognized as being unlike those of the more common miliary infections but suggestive of a reticulosis. The pathological lesions of chronic brucellosis were undoubtedly those of a granuloma—an infective reticulosis. Most authors had described pneumonitis and other pulmonary lesions as infrequent occurrences in brucellosis. However, Dr. Colebatch said that he had been able to discover in the literature only one example of miliary lesions in the lungs of a patient with brucellosis; in that case the patient had also had miliary granulomata in the bone marrow (Stettbacher and Wegmann). Dr. Colebatch then raised the question whether the pulmonary lesions in the case just presented were due to brucellosis.

A third point of interest was the value of the blood sedimentation rate. In uncomplicated brucellosis that was usually normal, and it had been stated to be of no diagnostic assistance. Nevertheless, in the case under discussion the raised sedimentation rate was the main finding that had led one to persevere in search of a diagnosis. Amaniera,

from a study of the test in 50 cases in Italy, had found that a normal sedimentation rate in association with pyrexia carried a good prognosis, but that a raised sedimentation rate with a normal temperature indicated that the patient was likely to relapse.

Finally, Dr. Colebatch raised the question of treatment. The boy presented had been treated with bed rest and aureomycin, which had been followed by a rapid fall in the sedimentation rate, diminution in the size of the spleen, and a gain of four pounds in weight, but no perceptible change in the radiological signs. One week after he left hospital the sedimentation rate had risen again to 30 millimetres in one hour. It appeared that terramycin was now regarded as the drug of choice for brucellosis. Dr. Colebatch wondered whether that would be sufficient to achieve a cure, or whether it would also be necessary to follow a *régime* of carefully graded steps in returning the patient to normal activities, as one followed in treating a patient with tuberculosis or rheumatic infection.

DR. W. J. STEVENSON said that a positive skin test result with brucellin was specific but did not differentiate past from present infection. After a brucellin skin test there was a rise in antibody titre which might last for several months and confuse the diagnosis of active infection. The condition of brucellosis was rare in Victoria even amongst slaughtermen, 50% of whom yielded positive Coombs test findings.

Dr. Perry assured Dr. Stevenson that the brucellin skin test had been carried out after blood had been taken for agglutination.

MR. E. FRENCH said that the rising prozone and Coombs test titres were very suggestive of brucellosis. Lesions could be widely disseminated in brucellosis, and liver biopsies performed at the Walter and Eliza Hall Institute showed granulomata. Mr. French then demonstrated a microscopic section of a liver biopsy from a patient with brucellosis. Circumscribed granulomata were visible. As that type of lesion was found in the liver, there appeared no reason why miliary granulomata could not occur in the lungs as well. Liver function tests might be informative in the case under discussion. Mr. French stressed the importance of making every effort to isolate the organism. Cultivation was difficult, as the organism was a strict aerobe and required an oxygen concentration of at least 10%. Additives to the blood culture medium could be used to destroy white blood cells and thus aid the growth of the organism.

DR. H. G. HILLER quoted Harvey, of New York, as having described two cases of proven brucellosis with miliary lung lesions.

DR. H. MCLORINAN, quoting Dr. Featonby, of the Health Department, said that Victorian milk was teeming with brucella. Many unrecognized cases of brucellosis must be present in the community.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

AN ERROR IN TREATMENT.[1]

[*The Australian Medical Journal*, 1876.]

"THE following is reported from the *Maryborough and Dunolly Advertiser*:

'It was reported to the physician of one of our Melbourne hospitals the other day, that there was a man lying in one of the wards in a comatose condition. The nurse declared that he had been insensible for twenty-four hours, and she had tried in vain to rouse him. The doctor said that it was probable that the patient was under the influence of some powerful narcotic; perhaps he had taken a large dose of laudanum. He said it was imperatively necessary that the unfortunate man should be resuscitated at once by some powerful stimulant. Accordingly he directed two of his assistants to take a strong galvanic battery, while the nurse stopped for a few moments in the laundry. When they reached the man's bedside, they placed the battery on the floor, and, baring the patient's ankle, they wrapped the wire around it. When everything was ready they turned on the current full head. A second later the prostrate form of the patient bounded about four feet in the air, and as it came down upon the bed, a second shock sent it up again, the patient meantime exclaiming: "Oh, murder, murder-r-r-r-r! Grashus! Let up on that. Another one of them'll kill me! O merciful Moses! Don't do that again." When he came down the fourth time the doctors turned off the current, with the remark that they thought that would be about enough. Then one of them asked the patient how he felt, and attempted to feel his pulse. But the patient, furious with rage, said: "You diabolical scoundrel, what d'ye mean by hitching that thing to me in that manner, say?" "Now, be calm", said the doctor, "it's all right, you'll be better directly." "But it isn't all right. I've a mind to knock your head off for blowing me up with that infernal machine. What d'you do it for, anyway?" "My friend, don't excite yourself", said the doctor, "you've been in a very bad way, and we ran the current through you to bring you back to life." "Bring me back to life! I was no more dead than you were." "Now keep cool. You have been unconscious for twenty-four hours. Narcotic poisoning, no doubt. We have saved you from an early grave. It was the closest shave. It was, upon my honour." "Well, well, if this don't beat all the ———. You took me for the man in Ward 3. Why, I'm one of the new wardsmen, and I lay down on this bed for a nap. The fellow you're after is over yonder. An early grave! Well, now I have heard foolishness in my life, but this takes the rag right off. And I give you warnin' that if you come about yer with that apparatus again, trying experiments on me, I'll wrench your brain-pan for you." Then the doctor moved off in search of the right man, while the keeper went out to hunt a dog to kick in order to relieve his feelings.'"

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

BRAIN SURGEON: THE AUTOBIOGRAPHY OF WILLIAM SHARPE.

SIR: The review in THE MEDICAL JOURNAL OF AUSTRALIA (September 5, 1953) of "Brain Surgeon" by William Sharpe brings forward the subject of lumbar puncture in those newborn infants showing evidence of intracranial trauma. All neonatal pædiatricians will, I think, agree that this author's contention that all cases of cerebral palsy can be prevented by lumbar puncture in the first week of life is unfortunately far from being correct. The worst cases are not due to hæmorrhage, anoxia often producing much worse results.

I would, however, put forward a plea that all newborn infants who show evidence of intracranial mischief should have a diagnostic lumbar puncture performed. It is only by this measure that a diagnosis of the causative condition can be made.

Roughly speaking there are four main types of intracranial disorder: (i) intracranial hæmorrhage, (ii) cerebral œdema, with swelling of the brain, (iii) intrauterine cerebral anoxia, (iv) increase in amount of cerebro-spinal fluid usually associated with severe extrauterine anoxia. Often the types occur together, hæmorrhage being frequently associated with anoxia.

Generally speaking these different types produce common syndromes of either (*a*) the irritated type with cerebral cry, restlessness, head retraction, adducted thumbs, difficulty in feeding, twitching, convulsions or (*b*) the lethargic type with drowsiness, apathy and pallor. Unless a lumbar puncture is performed it is impossible to know which condition is present, and as the treatment obviously depends on the condition it is impossible without lumbar puncture to give the appropriate treatment.

In doing a lumbar puncture it is wise to use an autoclaved needle, as in the normal full-time baby, one finds very little cerebro-spinal fluid in the theca—frequently only a drop or two in the needle. In the normal premature infants of three to four pounds there is much more fluid outside the brain (as has been shown by Edith Potter) and it is normal in them to get several cubic centimetres, which may be yellow if the baby is jaundiced. According to continental workers the protein content of the cerebro-spinal fluid in prematures is 100 milligrammes per 100 cubic centimetres for the first four weeks.

Subdural hæmorrhage of any considerable size will rupture into the subarachnoid space and give xanthochromic or evenly blood-stained fluid. There is no doubt that blood in the subarachnoid space acts as an irritant and produces

an aseptic meningitis, somewhere about the third day. This is the reason why the signs often exacerbate at this time.

When lumbar punctures are done daily, the much greater amount of fluid yielded at this time and the marked dilution of the blood are evidences of this.

The rise in intracranial tension and the increased protein of the cerebro-spinal fluid militate against the absorption of the cerebro-spinal fluid and so tend to keep the tension high. Repeated lumbar puncture as a therapeutic measure is definitely beneficial, at first daily and later at longer intervals till the tension becomes normal and the protein falls to 60 milligrammes per 100 cubic centimetres.

In the cases with an increased amount of cerebro-spinal fluid without hæmorrhage lumbar puncture is both diagnostic and therapeutic and rarely needs to be done more than two or three times.

In cerebral œdema and intrauterine anoxia one expects negative cerebro-spinal fluid findings.

The place of subdural tap is more difficult to evaluate. Because it is technically easier it is sometimes preferred. When one realizes that anoxia causes a lowering of the blood prothrombin and has seen at post-mortem examination an instance where a film of blood has spread over the brain after the tap, one hesitates to do this test unnecessarily. Should one do a subdural tap only when indicated, as (*a*) when localizing signs are present or (*b*) in a case which is not showing the usual improvement at the end of the week, or should it be done in all cases of intracranial trauma? I would be grateful for expression of opinion on this point.

Yours, etc.,

33 Collins Street, Melbourne,
September 8, 1953.

KATE I. CAMPBELL.

A BLOODLESS OPERATION FOR PARONYCHIA.

SIR: I have read with interest Dr. Kinsella's recent article on the treatment of paronychia and agree with him on the choice of the site for puncture.

I have found a sharp carbolized hard wooden point more satisfactory than the eye end of a surgical needle. After the puncture the avoidance of ointments and the application of an antiseptic dressing such as *Lotio Hydrargyri Perchloridi* 1:4000 is advisable along with the use of the appropriate antibiotic, hot finger baths and X-ray therapy.

In very early cases I have found, time and time again, that a suberythema skin dose of X-ray therapy of half-value layer of approximately 3·0 millimetres aluminium will very often abort the attack, especially if accompanied by the use of an antibiotic; a number of my colleagues can testify to the value of this procedure. Dr. Kinsella did not mention this method. By the method described by him, it is always easily possible to remove the proximal portion of the nail in cases where the pus had spread under it without resorting to the Kanavel operation or removal of the whole nail plate, which is sometimes done. A nerve block renders the procedure completely painless.

Yours, etc.,

170 St. George's Terrace, Perth,
September 5, 1953.

J. M. O'DONNELL.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Parramatta.

THE Post-Graduate Committee in Medicine, in conjunction with the Central Western Medical Association, will hold a week-end course at Parramatta District Hospital on Saturday and Sunday, October 17 and 18, 1953. The programme will be as follows:

Saturday, October 17: 2 p.m., registration; 2.30 p.m., "The Present-Day Management of Ante-Partum Hæmorrhage", Professor Bruce T. Mayes; 4 p.m., "The Present-Day Management of Toxæmia of Pregnancy", Professor Bruce T. Mayes.

Sunday, October 18: 10 a.m., "The Diagnosis and Management of Epilepsy in General Practice", Dr. K. B. Noad; 11.30 a.m., "Dermatological Out-Patient Clinic in 'Kodachrome': I", Dr. Adrian Johnson; 2 p.m., "Common Disorders of the Nervous System and Their Treatment", Dr. K. B Noad; 3.30 p.m., "Dermatological Out-Patient Clinic in 'Kodachrome': II", Dr. Adrian Johnson.

The fee for the course will be £3 3s. Those wishing to attend are requested to notify Dr. C. A. McDermott, Course Supervisor, Central Western Medical Association, 8 Carlton Street, Granville.

Week-End Course at Newcastle.

The Post-Graduate Committee in Medicine, in conjunction with the Central Northern Medical Association, will hold a week-end course at the Royal Newcastle Hospital on Saturday and Sunday, October 10 and 11, 1953. The programme will be as follows:

Saturday, October 10: 2 p.m., registration; 2.15 p.m., "Cardiac Failure", Dr. G. V. Hall, Dr. H. M. Windsor, Dr. D. Stuckey.

Sunday, October 11: 10 a.m., "Congenital Cardiac Defects", Dr. G. V. Hall, Dr. H. M. Windsor, Dr. D. Stuckey; 2 p.m., (*a*) "Coronary Occlusion", (*b*) "Cardiac Arrhythmias", introduction by Dr. G. V. Hall, followed by general discussion.

Fee for attendance at the course will be £3 3s. Those wishing to attend are requested to notify Dr. H. W. Rundle, Honorary Secretary, Central Northern Medical Association, Chatham Road, Broadmeadow, as soon as possible.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR OCTOBER.

Week-End Course at Horsham.

A WEEK-END COURSE will be held at Horsham on October 10 and 11 with the following programme:

Saturday, October 10: 2.30 p.m., Dr. Douglas Duffy, "Urological Problems"; 8 p.m., Dr. Donald Lawson, "Prolonged Second Stage of Labour".

Sunday, October 11: 10 a.m., Dr. Roderick Andrew, "Management of the Dyspeptic".

Enrolments should be made with Dr. A. L. Bridge, Lister House, Horsham. Fees are at the rate of 15s., payable to the Melbourne Permanent Post-Graduate Committee, 394 Albert Street, East Melbourne, but those who have paid an annual subscription to the committee may attend without further charge.

Lecture at Flinders Naval Depot.

A lecture will be given at Flinders Naval Depot at 2.30 p.m. on Wednesday, October 14, by Dr. James Guest. This is by arrangement with the Royal Australian Navy.

Course at Bendigo.

The course originally scheduled for Bendigo for October has been postponed till November 21, so that Dr. Douglas Guthrie, M.D., F.R.C.S., of Edinburgh, may be present to lecture on the history of medicine. His subject will be "Witches and Witchdoctors".

Obituary.

FREDERICK ARTHUR MAGUIRE.

DR. E. S. MEYERS writes: The death of Dr. F. A. Maguire reported in the journal, September 5, 1953, calls to mind the many medical officers of the Australian Army Medical Corps, Third Division, First Australian Imperial Force, who have passed away.

In addition to Colonel Maguire, A.D.M.S., there were his D.A.D.M.S., Major Vickers, and the following members of the Eleventh Field Ambulance—Colonel Chambers, Major Brummitt, Captain Ewing Thomson, Captain Faithfull, Captain Frank McCallum and others.

In France Colonel Maguire became A.D.M.S. of the Third Division, and was responsible for the medical arrangements during the advance of the division in 1918. After the Armistice, when things were quieter, he paid each of his field ambulances a visit on various occasions. On one occasion when driving back from the field ambulance to headquarters, he remarked to the officer driving him: "You know, Captain ————, I enjoy visiting the Eleventh Field Ambulance because they all tell you exactly what they are thinking."

After the war I came to know him much better by meeting him at various conferences and meetings of the College of Surgeons, and in 1932 he gave a lecture for the Queensland Division of the College entitled "Pelvic Repair Operations and Their Anatomical Repair".

On the occasion of this visit to Brisbane he visited the newly inaugurated Anatomy School in William Street, and dissected before an audience of the various specialists of the day the female pelvis. The dissection was beautifully done and illustrated the various points that he had elaborated in his excellent text-book, "The Anatomy of the Female Pelvis", which book is used in the University of Queensland as a text-book for medical students.

The lectures that he gave in Adelaide quite recently were masterpieces of gynæcological wisdom, and all students and practitioners would be the better off for reading them. He was no doubt a very great teacher of gynæcology.

Medical Prizes.

AMERICAN DERMATOLOGICAL ASSOCIATION PRIZE ESSAY.

THE American Dermatological Association is again offering a series of prizes for the best essays submitted for original work, not previously published, relative to some fundamental aspect of dermatology or syphilology. The purpose of this contest is to stimulate investigators to original work in these fields. Cash prizes will be awarded as follows: five hundred dollars, three hundred dollars and two hundred dollars for first, second and third place, respectively.

Manuscripts typed in English with double spacing and ample margins as for publication, together with illustrations, charts and tables, all of which must be in triplicate, are to be submitted not later than December 1, 1953. The manuscripts should be sent to Dr. J. Lamar Callaway, Secretary, American Dermatological Association, Duke Hospital, Durham, North Carolina. Those which are incomplete in any of the above respects will not be considered. Competition in this prize contest is open to scientists generally, not necessarily to physicians. Further information may be obtained by writing to the Secretary of the American Dermatological Association.

The next annual meeting of the American Dermatological Association will be held from April 13 to 17, 1954, at The Greenbrier Hotel, White Sulphur Springs, West Virginia.

AMERICAN COLLEGE OF CHEST PHYSICIANS STUDENT PRIZE.

THE Board of Regents of the American College of Chest Physicians offers three awards to be given annually for the best original contribution, prepared by any medical student studying for the degree of doctor of medicine, on any phase relating to the diagnosis and treatment of chest disease.

The first prize will consist of a cash award of $250 and a certificate. The second and third prizes will be certificates of merit. The essay award is open to all medical students in accredited medical schools throughout the world.

The winning contributions will be selected by a board of impartial judges and will be announced at the twentieth annual meeting of the American College of Chest Physicians to be held in San Francisco, California, June 17 to 20, 1954. All manuscripts become the property of the American College of Chest Physicians and will be referred to the editorial board of the College journal, *Diseases of the Chest*.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 22, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	2(2)	..	..	2(2)	..	..	..	4
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	4(4)	..	..	..	2	..	6
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	1(1)	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1(1)	4(4)	..	..	..	2	..	..	7
Diphtheria	2(1)	1(1)	4(4)	1(1)	2(2)	..	..	..	10
Dysentery (Bacillary)	..	1(1)	..	..	..	1(1)	..	..	2
Encephalitis	1(1)	..	..	..	..	2(2)	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	1	..	..	..	..	..	..	..	1
Infective Hepatitis	..	19(4)	..	..	10(4)	..	..	..	29
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	1(1)	..	..	..	..	1	..	2
Leptospirosis	..	..	1(1)	..	..	..	..	..	1
Malaria	..	1(1)	..	..	1(1)	..	..	..	2
Meningococcal Infection	3(3)	3(1)	..	1	..	..	..	..	7
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	9(6)	1	4	..	..	..	..	..	14
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	16(8)	1(1)	..	20(15)	..	..	..	37
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	12(5)	52(42)	6(6)	4(3)	2(1)	..	..	..	76
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	44(35)	15(8)	8(3)	6(5)	5(4)	4(3)	..	..	82
Typhoid Fever	2	2(2)	2(1)	..	..	..	..	..	6
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

for consideration. The College reserves the right to invite the winner of the first prize to present his contribution at the annual meeting.

Applicants are advised to study the format of *Diseases of the Chest* as to length, form and arrangement of illustrations to guide them in the preparation of the manuscript. The following conditions must be observed: (i) Five copies of the manuscript typewritten in English (double-spaced) should be submitted to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, not later than March 15, 1954. (ii) The only means of identification of the author shall be a motto or other device on the title page and a sealed envelope bearing the same motto on the outside enclosing the name and address of the author. (iii) A letter from the dean or chairman of the department of medicine of the medical school certifying that the author is a medical student studying for the degree of doctor of medicine and that the contents represent original work.

Further inquiries may be directed to the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, who will forward them to the local representative of the College.

Medical Practice.

REGULATIONS RELATING TO DANGEROUS DRUGS AND SPECIFIED DRUGS IN VICTORIA.

THE following has been added to the Schedule of Specified Drugs in Victoria: Phenylbutazon, whether butazolidin or by any other name, and all preparations thereof.

Deaths.

THE following deaths have been announced:

SHORTER.—Herbert Leopold Ashton-Shorter, on September 5, 1953, at Epping, New South Wales.

YOUNG.—James Hannibal Young, on September 6, 1953, at Mount Hawthorn, Western Australia.

YOUNG.—Kevin Young, on September 10, 1953, at Melbourne, Victoria.

GARDINER.—Leslie Andrew Gardiner, on September 16, 1953, at Sydney.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Bell, James, M.B., B.S., 1930 (Univ. Sydney), 17 Pound Street, Lismore, New South Wales.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Carter, Ian Rowland, M.B., B.S., 1953 (Univ. Sydney); Johnston, Robert Winstanley, M.B., B.S., 1953 (Univ. Sydney); Mackey, Kenneth Halford, M.B., B.S., 1953 (Univ. Sydney); Weinrauch, Ludovic, M.B., B.S., 1953 (Univ. Sydney); Whitling, William Dumas, M.B., B.S., 1953 (Univ. Sydney); Vickers, Brian Hugh Philip, M.B., B.S., 1952 (Univ. Sydney); Byrne, John Justinian, M.B., B.S., 1950 (Univ. Sydney); Harmelin, Elkan, registered in accordance with the *Medical Practitioners Act*, 1938-1950, Section 17 (1) (*c*); O'Dea, Bettine Frances, M.B., B.S., 1946 (Univ. Sydney).

The undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Stoutjesdijk, Albertus Diedenk Johan, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), 6A Colyer Street, Centennial Park, South Australia.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Bentley, Alan, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Brentnall, George Rex, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Nankivell, Shirley Dawn, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Wighton, Dugald Craven, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Hoff, Lothar Clemens, M.B., B.S., 1953 (Univ. Adelaide); Linn, John Malcolm, M.B., B.S., 1953 (Univ. Adelaide); Pfitzner, Murray Aikland, M.B., B.S., 1953 (Univ. Adelaide); Smith, Rayner Whitmore, M.B., B.S., 1953 (Univ. Adelaide); Whitehouse, Joseph Chenoweth, M.B., B.S., 1953 (Univ. Adelaide).

Medical Appointments.

Dr. T. L. McLarty and Dr. D. O. Crompton have been appointed honorary ophthalmologists at the Royal Adelaide Hospital.

Dr. D. W. Brummitt and Dr. M. C. Moore have been appointed honorary assistant ophthalmologists at the Royal Adelaide Hospital.

Diary for the Month.

OCT. 2.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates.
OCT. 6.—New South Wales Branch, B.M.A.: Council Quarterly.
OCT. 7.—Victorian Branch, B.M.A.: Branch Meeting.
OCT. 7.—Western Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10*s*. *per annum* within America and foreign countries, payable in advance.